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THE TONSILS IN CHILDHOOD*

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The tonsils have attracted many investigators, but the results of the investigations are unsatisfactory and often contradictory. Connected by many in a very direct way with local disease elsewhere as well as with systemic disturbances, it is especially necessary because of the incompleteness of our knowledge that we should have our attention directed at times to their functional side lest we rashly assume that the tonsils are to be regarded as a constant menace to health. It will therefore not be out of place to restrict my remarks chiefly to the relation of their function, so far as we at present know it, to disease, and along these lines to indicate the general principles that should underlie our treatment, rather than to specify the particular treatment adapted to any special variety of disease which they present.

We may regard disease as a condition of disturbed or imperfect function or of the results of disturbed function. The day is past when pathology was a study of morbid anatomy; it is now to a large extent a study of morbid physiology. Therefore, to observe correctly and record accurately the signs and symptoms of the disease of any organ, we must necessarily have a clear picture of its physiologic function. Failing to get such a picture, we must fall back on such anatomic facts as bear on the function. Since disease, though local in its outward manifestations, is systemic in its effects, we again require to fall back on anatomy and physiology so as to correlate correctly the disturbance of function with pathologic and bacteriologic changes produced elsewhere. Only thus can we hope to gain an intelligent grasp of ultimate conditions.

So it appears to me that in approaching the subject of the tonsil, shrouded as it is with so much uncertainty, it would be well to emphasize such points in its anatomy and physiology as appear important for the clinician to have recalled, and especially as some of these are too often overlooked or misinterpreted. Many of our errors in local treatment have resulted from failure to grasp some fundamental fact in its anatomy or physiology. Some of our failures can be traced to an inability to correlate intelligently the relation of systemic results to local causes or local results

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to systemic causes. It will be especially necessary for me to emphasize these anatomic and physiologic observations, since I shall present to you some conclusions which I have been forced to draw from them, bearing on tonsillar disease especially relevant to the pediatrician. Further, I shall dwell on these observations since I believe the clearing up of many of the enigmas associated with the pharyngeal tract necessitates not only a clear conception of the present state of our knowledge, but a cooperation of pediatrician with laryngologist.

The ease with which the palatine tonsil is observed and the association which it has in the minds of many with all kinds of diseases, lead often to a self-satisfying yet inaccurate diagnosis. Forgetful of its intimate relation to pathologic processes in the nose and nasopharynx, and of its frequent association with systemic disturbances, we too readily diagnose as a primary pathologic condition what is in reality a secondary pathologic result. In a tonsillar enlargement we often erroneously see an active pathologic process instead of a hyperfunctionating organ.

My remarks apply particularly to the palatine tonsils, usually referred to as "the tonsils," and to the pharyngeal tonsil lying in the nasopharynx, enlargements of which we loosely speak of as adenoids. Anatomically we possess a fairly clear picture of their structure and a fairly accurate knowledge of their phylogenetic and ontogenetic history, especially of the palatine tonsils. Physiologically so much is even yet obscure that in the minds of many this lack of knowledge is taken to mean that we must regard the tonsil as of no importance. To this some have added a baneful association with all kinds of diseases, and so they reach that ultranegative stage in which the tonsil becomes an actual menace to health. Gradually, however, evidence is accumulating which is helping to a clearer understanding.

On the *anatomic* side the facts I wish briefly to emphasize are:

1. The palatine tonsil is present in all mammals, with one or two noteworthy exceptions in rodents. Thus the rat and guinea-pig have no tonsils. I can confirm the opinion of other investigators in regard to the rat, that not only are no tonsils present, but there is no sign in the embryo of any tonsillar anlage. The significance of their absence has so far not been explained. From a comparative study it would appear that the size of the tonsil bears no relation to the species, but apparently has relation to the size of the animal.[1] The anatomic location of the tonsil is such that no matter how it be concealed by folds of mucous membrane, it always retains a communication with the pharynx. I have been much impressed with this fact from observations made in my laboratory during the past winter, especially in carnivora. In this genus the tonsil is often so protected by folds as to be invisible from the mouth, no doubt influ-

1. Poole, C.: Thesis for M.A. degree, Northwestern University, 1911.

enced by the character of the food devoured. But there always exists a channel of communication with the pharynx, This is well seen in the lion, in which the tonsil lies in an elliptical sac of considerable size in the lateral pharyngeal wall, occupying but a portion of one side. The sac communicates with the pharynx by a relatively small opening, and is so placed that during certain movements of the pharynx the contents can be expelled into the back of the mouth. Therefore, comparative anatomy suggests in the varied relations which the tonsil bears to the pharynx, the necessity of a direct communication between the tonsil and pharynx along which secretion or excretion may pass from the tonsil to the pharynx.

2. In all mammals the tonsil does not develop like a lymph-node from a plexus of preexisting lymph-vessels in the mesothelium. It develops as an ingrowth of endothelium from the second branchial pouch, and later lymphoid tissue appears in the mesothelium around these ingrowing buds. In origin it comes into line with the thymus and the thyroid. Therefore developmentally the tonsil is not a lymph-node.

3. When we examine the life history of the tonsil we find that it appears early in embryonic life (fourth month), reaches maturity at the end of the first year of infancy, and at or about puberty tends to diminish in size. In the adult the reticular tissue and follicles are more or less atrophied. From these facts we may say that there is good ground for the belief that the physiologic activity of the tonsil is of greatest importance in early life.

On the *physiologic* side our knowledge of the tonsil is extremely meager. We know that in its deeper tissue we have lymph-nodules, some with germ centers showing the proliferation of lymphocytes which pass either internally into the lymphatics or externally into the crypts of the pharynx, constituting part at least of the so-called salivary corpuscles. As Stohr[2] pointed out, the exudation of leukocytes is physiologic and not inflammatory, since it is observed in healthy children and at the end of embryonic life. In healthy individuals the exuded leukocytes do not succumb at once, but mix with the alkaline mouth secretions, and according to Grober,[3] on a warm stage show ameboid movements. But what is their further action? Do they act as phagocytes or do they give up enzymes? Metchnikoff[4] has no doubt about the answer:

> The tonsils and other lymphoid organs are traversed by an enormous number of leukocytes which execute a kind of migration toward the cavities containing microorganisms. It is evident that we have here a process of phagocytic defense. When we remove a particle of mucus from the surface of the tonsils of a person

2. Stohr: Zur Physiologie der Tonsillen, Virchows Arch. f. path. Anat., 1884, xcvii, 211; Biol. Centralbl., 1882.

3. Grober: Tonsillen als Eintrittspforten für Krankheitserreger, Klin. Jahrb., 1905, xiv, Part 6.

4. Metchnikoff: Immunity in Infective Diseases, 1905, Cambridge Univ. Press, p. 415 and 428.

in good health we always find that it contains leukocytes, especially microphages, filled with microorganisms of all kinds.

In further explanation of the defensive properties he continues:

The buccal cavity has a very rich microbial flora. It is astonishing that in spite of this state of things, wounds in the mouth heal very rapidly, and operations on the buccal cavity done with insufficient or no aseptic precaution do not, in the great majority of cases, set up infective complications of the slightest importance. It is often asked how under these conditions does the mouth defend itself against the vast number of formidable microorganisms? Powerless as an antiseptic, the saliva fulfils an important function in ridding the mouth of microorganisms in a mechanical way, cleansing and washing them away, mechanically to the exterior, more frequently into the stomach. In addition to the direct mechanical part played by the saliva, it performs a very important indirect function. This fluid contains microbial products and diastases, and is capable of exciting in the leukocytes a positive chemiotactic activity. The influence of the saliva on the afflux of the leukocytes must be regarded as an act important for the protection of the buccal cavity, and it is probably due to this attraction of leukocytes that lesions of this region heal so quickly. The leukocytes are very numerous in the glands of the mouth and the tonsils always supply large quantities of them.

Before leaving this phase of the subject I would add that from observations recently made at the Wesley Hospital I have been led to believe that in systemic diseases we may have this exudate very considerably modified, and according to Grober[3] it may disappear during diseases which accumulate leukocytes within the body.

But from the activity of the tonsil in fetal life, as shown by the fact that at birth the crypts and tonsil lymph-sinuses are found to contain lymphocytes, it would appear that the exudation from the tonsil cannot have only a relation to bacterial invasion of the pharynx. Its greatest activity being during developmental life, would suggest some additional function. From our knowledge of leukocytes, and with the experiments of Kroeppe[5] in mind, I cannot but feel that we have in the tonsil an organ combating some constituent of metabolism which may appear in the lymph-channels or in the nasopharynx, a constituent which if passed into the blood-stream would be hurtful to the individual. When this product of metabolism is arrested or disappears the leukocyte activity ceases and the gland is converted into ordinary connective tissue, the germ center to be the last to go. It has been suggested that tonsillar metabolic processes are connected with development of the teeth.[6] This may be so; but let us not forget that there are other complex processes going on in the upper respiratory and alimentary tracts in childhood. Under the circumstances it would be well to wait for further observations.

From the above anatomic and physiologic data I have drawn the following *conclusions:*

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5. Kroeppe: See Gulland, Development of Lymphatic Glands, Jour. of Path. and Bact., 1894, p. 476.

6. Wright: Boston Med. and Surg. Jour., May 20, 1909.

1. Since comparative anatomy shows that in all cases a communication with the pharynx has been preserved, it would appear that intimate relationship with the pharynx is a necessity of tonsillar activity.

2. The tonsillar activity is to be regarded as most active during developmental life. There is no evidence that in man it should be regarded as a recessive organ.[7]

3. The tonsil cannot be regarded as merely a lymphatic node. Though it presents microscopically adenoid tissue, yet its developmental history and its physiologic activity at least make us pause before drawing such a conclusion. It is just this presumption that has led many to a hasty and unnecessary enucleation. The statement some have made that since the tonsil is only a lymphatic gland its activity can be readily replaced by other lymphatic structures is of very doubtful merit even so far as lymph-nodes are concerned, and erroneous so far as the tonsil in the child is concerned.

4. We have every reason to believe that the tonsil plays an important rôle in the complex changes which occur at the upper end of the alimentary tract. The argument that no one has seen any local or systemic deficiency following removal of tonsils in childhood does not satisfy me, since we have not sufficient observations based on complete enucleations. Complete enucleation is at all times difficult, as one can see on examining the deeper anatomic appearance of an enlarged tonsil.[7] Further, we cannot know what damage may be done till we have a clearer conception of its function. Let the pediatrician watch judiciously cases of tonsil removal and from him and not from the laryngologist I will expect to hear before long of some observation which may be more than a mere coincidence between tonsillar enucleation and systemic effects. Already we have a few suggested.

As an outcome of these anatomic and physiologic considerations I have been led to believe that in the child we are dealing with an organ which functionally differs considerably from the same organ in the adult. I therefore distinguish in the life-history of the tonsil *two distinct periods:*

(*a*) Before puberty — period of functional activity.

(*b*) After puberty — when it persists chiefly as an aggregate of lymph-nodules which tend to atrophy.

This point of view very considerably modifies my views in regard to tonsillar diseases and their treatment. In the child it should be conservative, so far as possible, and the younger the child the more conservative; in adults let it be radical whenever necessary.

What is a normal tonsil in childhood?

When we turn to anatomic authorities for assistance in answering this question we are met with great difference of opinion. Few consider the

7. Wilson, J. G.: Some Anatomic and Physiologic Considerations of the Faucial Tonsil. Jour. Am. Med. Assn., May 26, 1906.

question of size in the child as distinct from the adult, and so the figures are very divergent. As a fairly reliable basis I may quote from Cunningham[8] in regard to the size of the normal tonsil in early life: length, 20-22 mm. (1 inch); breadth, 18-20 mm. (three-fourths inch); thickness, 12-15 mm. (one-half inch). These, I presume, are post-mortem figures, though he does not say so.

Leaving the anatomist and coming to the laryngologist, the best figures I know of are given by Gleason[9] with regard to a child of 6: length, 1¼ inches; breadth, three-fifths to 1 inch; thickness, three-fifths to 1 inch. Again I do not know how he arrived at these figures, though it is evident from his remarks that he regards the tonsil as reaching its maximum size about that age.

Without desiring to discuss the relative value of these figures I should like to point out the following:

1. It is impossible to estimate the thickness of the tonsil at any post-mortem, especially after a chronic illness. These bodies shrink very considerably during the last stages of disease, as well as after death, as a result of their great vascularity.

2. The most reliable data have been got when the tonsil has been removed immediately after sudden death.

I am not aware of sufficient figures derived during life from young healthy children which would establish a normal. My observations would agree more with those of Gleason than with those of Cunningham, though if Cunningham's observations were post mortem and Gleason's observations were made during life or immediately after death in a brief illness, the discrepancies are not so marked. I believe that in a child the healthy tonsil projects beyond the anterior pillar of the fauces; that at the age of 6 to 12 it is not normal to find it concealed behind the pillar. Considerable variation in this projection is consistent with health, not only at different periods in childhood, but even in a child. I do not believe that we can assign to a body so vascular and so responsive in childhood a definite depth or projection beyond the pillars below or beyond which it is abnormal.

We pass now from the résumé of anatomic and physiologic data to an application of our deduction from these data to some varieties of tonsillar disease. All are agreed that if the tonsil be diseased it should be removed. To this I would add that if so responsive a body appears functionally inactive and a probable source of disease its treatment or removal ought to be considered. But it is sometimes not so simple a matter to recognize a tonsil which demands removal because of disease. It is not so easy to say whether it is diseased primarily or whether its pathologic appearance

8. Cunningham, O. T.: Text-Book of Anatomy, New York, 1909, p. 1036.

9. Gleason, E. B.: Diseases of Nose, Throat and Ear, Philadelphia, 1910, p. 181.

may not be secondary to disease elsewhere, either in its immediate neighborhood or arising from systemic causes. In children it may be necessary to consider whether there is here a *hyperfunctionating* or *hypofunctionating* organ. Many large tonsils have been found to differ little if at all from a normal tonsil. Many tonsils macroscopically small and innocent looking have been found microscopically to contain numerous foci of infection.

Very little attention has been paid to the small palatine tonsils in children. As an outcome of my anatomic and physiologic beliefs, and as a result of observations in the clinic, I feel that we ought to consider these more carefully than we do at present. In the present status of medical practice they come more usually under the observation of the pediatrician than of the laryngologist, for such has been the trend of our teaching that in children we consider only such to be locally diseased as are enlarged or show signs of recurring inflammation. Such tonsils appear in children as small pale structures often concealed behind the pillars of the fauces, flabby in consistence and with wide mouthed crypts, often with whitish looking epithelium at their entrance. Microscopically I have frequently noted hyaline degeneration and minute foci of inflammation within the tissue. Sometimes half the tonsil is atrophied and the other half shows a ragged granulating pharyngeal surface projecting beyond the pillars. These hypofunctionating bodies may be associated with general pharyngeal attacks of a more or less acute nature in which the tonsil appears to play a subordinate rôle. They are apt to be associated with glandular enlargement in the cervical region. But it is when we regard the tonsil less in its local than in its systemic potentialities that we will have our attention fixed more and more on the atrophic variety. In a child when considering the tonsil in its causal relation to systemic disease I should regard with much more suspicion a small tonsil than a tonsil remarkable for nothing but its size. I have removed in a child of 8 such a tonsil associated with mild pharyngeal attacks, for persistent slight temperature associated with recurring rheumatic attacks, and with satisfactory results.

The enlarged tonsil so frequent in early life may show great variation in size consistent with a healthy organ. A large tonsil in a child is not necessarily pathologic; a normal tonsil may project considerably beyond the pillars. In many systemic diseases, apart from any local affections, it becomes enlarged. Mere fluctuation in size in a child ought not to be regarded as a sign of disease and an indication for enucleation. Many children are constitutionally subject to such fluctuations and the treatment is general more than local. It is common knowledge that in systemic diseases we have such enlargements and few of us are now disposed to argue that this enlargement is a sign of focal disease. I would rather regard it as an indication of functional activity. We cannot judge of

the condition of a tonsil from its appearance during an acute attack, for not only in systemic disease but in disease elsewhere in the nasopharynx the tonsil may become secondarily affected and may be only manifesting a hyperfunctionating activity. It is so easy to look into a throat and find an inflamed area with enlarged tonsils and arrive at a too hasty conclusion in regard to the cause of the illness.

In children many of the symptoms attributed to enlarged tonsils, such as anemia, buccal respiration, snoring, suffocative symptoms, inframammary depression of the ribs, ill-developed thorax, and especially eustachian deafness, are usually due to concomitant adenoids. But there are certain well-defined symptoms which indicate when we may well interfere. In this respect I cannot do better than give the following excellent summation from two well-known laryngologists:[10]

> Chronic enlargement of the tonsils may then act injuriously in three different ways, namely, (*a*) by mechanically obstructing the food and air passages; (*b*) by maintaining a liability to frequent, often very painful attacks of inflammation within the glands themselves or in their immediate neighborhood, and by the chronic toxemia from the contamination of the blood circulating through the tonsil, causing the so-called "poor circulation" or lack of normally active tissue-metabolism so often seen in patients with enlarged tonsils; (*c*) by forming a perpetual source of danger from infection by various microorganisms, such as those of diphtheria or tuberculosis (page 169).

I have quoted this paragraph *in extenso,* though as my previous remarks indicated, I would make some reservation in regard to the last sentence. In enlarged glands I have never found tubercle primarily present; this, however, I will discuss later. It is, however, a daily experience that such tonsils are subject to repeated attacks of inflammation and so may be legitimately removed.

In discussing the relation of focal disease in the tonsil to disease manifesting itself elsewhere, it may be asserted that there is sufficient evidence to lead us to conclude that some such relationship exists. This is what we would expect when we reflect that no disease is absolutely local and many react deleteriously on a distant part which is physically weakened from some cause, constitutional or otherwise.

To narrow our remarks down to specific example we may refer to the relation of tonsillar disease to arthritis or endocarditis. As I have said elsewhere,[11] there is sufficient clinical evidence to lead us to believe that a focus of infection anywhere in connection with serous or mucous surfaces may result in arthritic or cardiac affections, and that the removal of such a focus is often followed by the amelioration of such affections. Investigation has demonstrated that streptococci derived from various sources may produce arthritis and endocarditis in rabbits. Further, it would appear that the channel of introduction is of minor importance.

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10. Semon and Watson: System of Medicine, Allbutt and Rolleston, Londen, 1908, p. 169.
11. Wilson, J. G.: Illinois Med. Jour., February, 1912, p. 221.

At present the evidence seems to be to suggest the association of various organisms with this relationship rather than a special microbe, but on this point I would refer to the recent work of D. J. Davis,[12] who has justly qualified himself to speak with authority.

It is to be expected that we should find such examples to abound in connection with chronic focal infections in the nose, mouth and pharynx, where various strains of streptococci are found in abundance. It must be admitted that a chronic focal infection in the tonsil may bear some causal relation to arthritic and cardiac affections. But the frequency of the focal infection and the infrequency of a concomitant or subsequent arthritic affection speak against the relative importance of such a seat and suggest that some other factor has to be taken into account. My present attitude in regard to the etiologic relation of the tonsil to endocarditis and articular rheumatism may be summed up in this, that if the tonsil be subject to recurring attacks of inflammation it is not alone the tonsil which is vulnerable. It makes all the tissue in the nasopharynx more susceptible to infection. We thus have a large area from its position exposed to microbial attacks. Thence comes the initial factor in the chain which may lead through unknown paths to distant pathologic processes

Primary tuberculous disease of the tonsil is rare; disease foci secondary to pulmonary disease is not uncommon, the general belief being that there has been infection from the sputum. It is interesting here to note that it is not the enlarged hyperfunctionating gland that usually becomes primarily tuberculous, but the small hypofunctionating organ. Many observers have found tubercle bacilli in the tonsil, apart from actual disease, but the finding of this bacillus in the tonsil is not to be wondered at and is not indicative of disease, for they may get there through its lymphatics which connect with the nasopharyngeal mucosa, as well as by in-wandering from the pharynx. When we consider the liability of the child to tuberculous invasion, and the anatomic structure and position of the tonsil exposing it to infection in a way that few other organs are exposed, then add to this the continual rupture of its pharyngeal walls from exudation of leukocytes, the wonder is not that tubercle bacilli are to be found, but that primary tuberculous tonsils are rare. If the soil were as favorable as the bacteria are common, tuberculous tonsils would be the rule, not the exception. In children the invasion from the nasopharynx of the cervical lymph-nodes is undoubted, but tuberculous disease is but one of many causes of enlarged nodes, and the tonsil is but one of many sources. When the tonsil is at fault, I believe it is more often the small hypofunctionating tonsil which is to be found. It is an everyday experience that enlarged cervical lymph-nodes are often associated with tonsillar disease and that removal of this focus of disease frequently

12. Davis, D. J.: Jour. Am. Med. Assn., July 2, 1910, p. 26.

causes the glands to reduce in size. But in a majority of cases in children to make the diagnosis that these glands are tuberculous secondarily to enlarged tonsils rests on insufficient ground. The infrequency of primary tonsillar tuberculous disease, the rapid growth of these glands, and their rapid disappearance after removal of the tonsils appear to me to speak against their tuberculous nature. Another frequently expressed statement is that there is a special and direct connection between the tonsil and the apex of the lung for tuberculous invasion. This appears to me to be disproved on anatomic grounds, for no such direct connection between the cervical lymphatics and pulmonary lymphatics at the apex exists.

Before enlarged chains of nodes are removed from the neck it should be regarded as imperative that the nose, nasopharynx and pharynx be carefully examined to ascertain a possible source from which these nodes have become infected. Sometimes as a result of such an examination the complete surgical removal of cervical lymph-nodes now objected to on general principles would become unnecessary.

In conclusion let me say that my endeavor has been, so far as our knowledge at present permits, briefly to point out the probable function of the tonsil to metabolic processes and to diseases in the upper respiratory and alimentary tracts, but especially at their junction in the pharynx. As knowledge advances there is constantly impressed on us the important and unexpected relation of this region to disease. For instance, let me refer to its relation to cerebrospinal meningitis and poliomyelitis. We shall err if we forget that the essential resisting power of this tract to infection lies in its tissue cells. Further, we shall add to this error if we concentrate our attention too much on leukocyte invasion or exudation to explain initial protection. Whenever from unhygienic surroundings or mode of life, it may be also from constitutional weakness, the tracts are exposed to influences which lower their vitality, the tonsils suffer for them and with them. When the mucous surfaces, particularly absorbent in the child, are exposed to irritation, the tonsil again suffers for and with them. To me the tonsil in a child protects these tracts, especially where the two come together in the nasopharynx, and is associated directly with their well-being. If this be so, I am called on to do everything to protect these bodies during the period of functional activity. Further, I am called on to remove them when I am convinced that on account of disease or loss of function they are not only a source of infection but are having an injurious effect on the structures around.

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A COMPARISON OF THE STREPTOCOCCI FROM MILK AND FROM THE HUMAN THROAT

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The present comparative study of throat and milk streptococci was undertaken with the object of establishing a method for distinguishing between organisms isolated from these two sources. Much confusion has existed as to the relationships within this genus. The introduction of the biometric study of the streptococci according to their reaction in various carbohydrate mediums by Gordon (1904-1905) and the subsequent extension of the study by Houston (1905-1906) seemed to give a method of solving this obscure problem. The English results have been obtained by the use of litmus added to the mediums and the reactions recorded as positive or negative according to the color of the litmus, without titration. If the mediums were not neutral at the start, it is obvious that they must have classified many organisms as fermentors which have only very slight fermentative properties and really belong to the non-fermenting group. Winslow has pointed out that "they include a much greater percentage of positive records than the American ones."

TABLE 1.—PERCENTAGE OF CULTURES GIVING MORE THAN 1.1 PER CENT. ACID AT 37 C.

Fermented.	Per cent. Throat Cultures.	Per cent. Milk Cultures.
Dextrose only	22.2	6.3
Dextrose and lactose.....	20.4	38.0
Dextrose, lactose and saccharose	33.1	57.7

TABLE 2.—PERCENTAGE OF CULTURES GIVING MORE THAN 0.5 PER CENT. ACID AT 20 C.

Fermented.	Per cent. Throat Cultures.	Per cent. Milk Cultures.
No fermentation	50.0	0
Dextrose only	40.0	32.5
Dextrose and lactose.....	1.8	31.0
Dextrose, lactose and saccharose	0	31.0

Winslow introduced the method of titrating the cultures with phenolphthalein as an indicator, and at present this procedure seems to give a substantial basis for fixing the relationships within this group.

We examined seventy pure strains of streptococci isolated from fresh specimens in our laboratory. Cultures taken from sore, inflamed, or

otherwise unusual throats have been grouped as "abnormal." The small number of cultures used renders our generalizations more or less tentative, and it is our intention to substantiate the work, using larger numbers. Quantitative acid production was studied in six carbohydrate mediums: dextrose, maltose, lactose, saccharose, raffinose and mannite. Two tubes of a 1 per cent. carbohydrate broth were inoculated[1] at the same time, one tube being placed at 20 C. and the other at 37 C. for seventy-two hours. Five c.c. were then drawn off with a pipet, diluted with 45 c.c. of distilled water and titrated against twentieth normal sodium hydroxid, using phenolphthalein as an indicator. Blank tubes were incubated with the

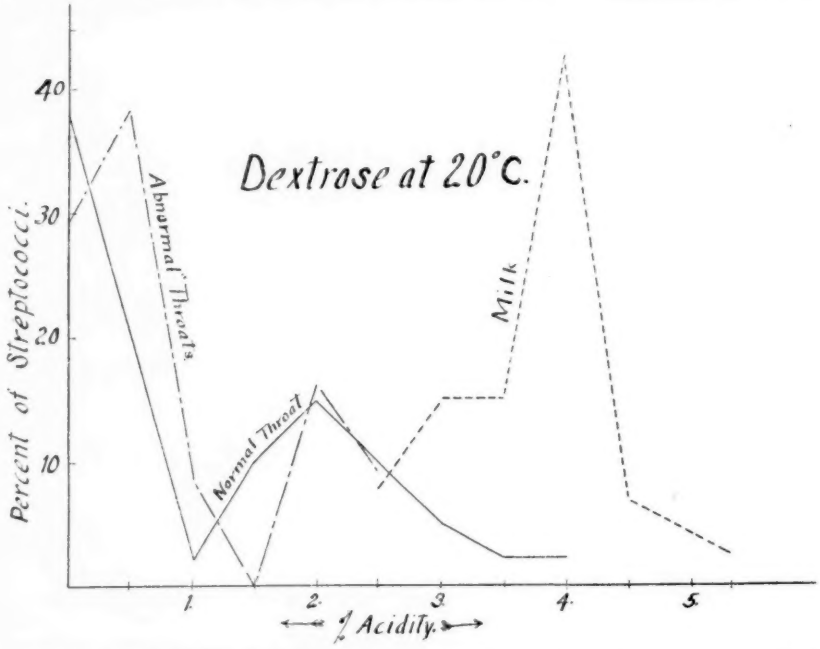

Chart 1.—Showing percentage of acidity and streptococci from abnormal throats, normal throats and milk cultured in dextrose at 20 C.

inoculated tubes and these were titrated at the same time. The difference between the blanks and the cultures was recorded as the amount of acid produced. We have omitted the maltose results in classifications tables, as the figures follow the lactose figures fairly closely. With the sugars that are fermented at all at 37 C., the first reaction mode was found to fall at or beyond 1.2 per cent. acidity expressed as normal acid, and we have used this as the dividing point between a positive and a negative reaction.

1. The inoculations were made by transfering two standard loops full of the growth found in the water of condensation on North's gelatin agar medium.

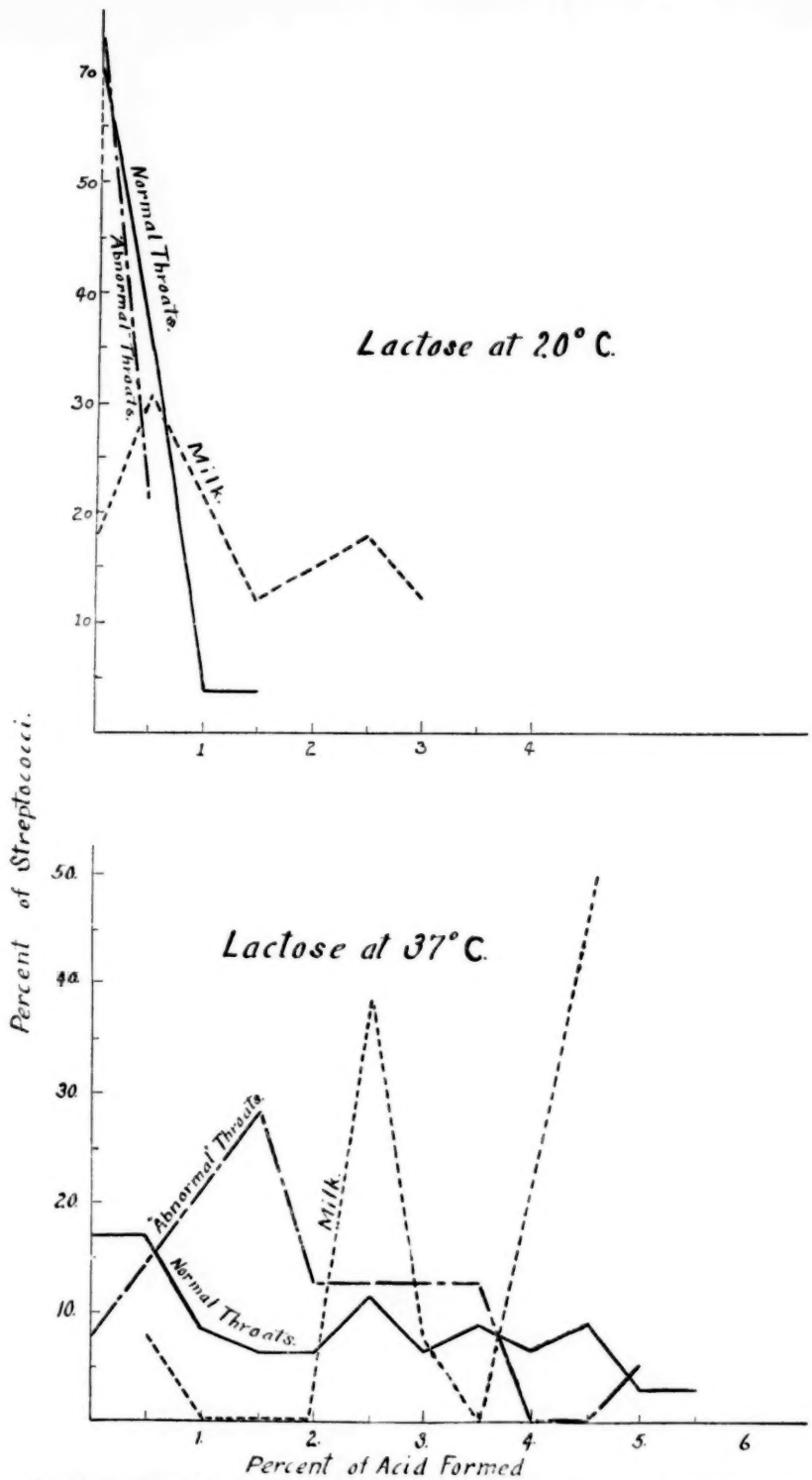

Chart 2.—Showing percentage of streptococci and acidity from normal and abnormal throats and milk cultured in lactose at 20 and 37 C.

On this basis, all but four cultures fermented one or more of the mediums used. It was found that 83 per cent. of these might be placed in three groups according to their fermenting capacity.

When incubated at 20 C. the first reaction mode falls at 0.5 per cent. acidity and on this basis the milk cultures fall into the same general groups as noted above, but the throat streptococci show 50 per cent. not fermenting at all, and 40 per cent. fermenting dextrose only.

It will be noted that the carbohydrates were attacked progressively in the order of the complexity of the molecular structure, the single sugar

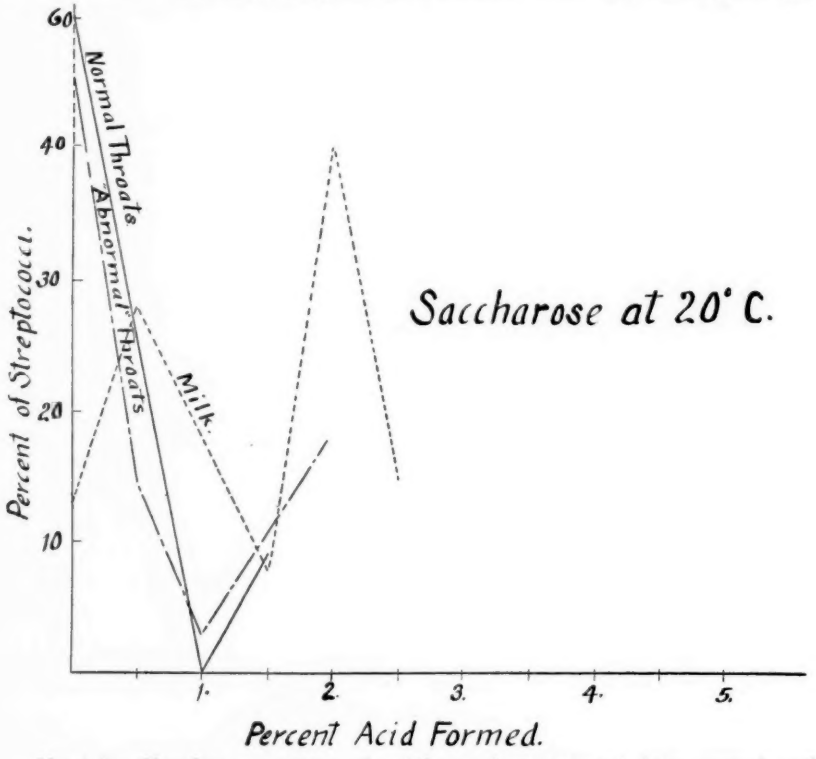

Chart 3.—Showing percentage of acidity and streptococci from normal and abnormal throats and milk cultured in saccharose at 20 C.

being most easily fermented, then the disaccharids, the trisaccharid and the alcohol being too complex to be used except by a very few cultures. It is seldom that a strain reacts with a sugar lower in the scale of fermentability while failing to attack all the simpler, more available ones, as is shown from a study of the tables at the end of this paper. Andrews and Horder suggested this grade of availability in 1906, referring to the phenomenon as "a set of gymnastic exercises in chemical decomposition." The colon group shows a similar metabolic relationship toward the carbohydrates but with them the size rather than the structure of the molecule

seems to be the controlling factor. Howe (1911) has aptly called this order of availability the "metabolic gradient." It suggests a very important advance in the biometric method.

A consideration of the reactions in the individual sugars brings out the following facts:

Dextrose: The normal throat strains seem to show the greatest fermentative properties, their mode coming at 6.0 per cent. acid when grown at 37 C. The milk cultures show a very specific reaction, all falling into a group giving from 3.5 per cent. to 5.5 per cent. acid. Reference to

TABLE 3.—STREPTOCOCCI ISOLATED FROM NORMAL THROATS

Sample No.	Long or Short Chain Form	Reaction	Percentage of Acid Formed in: Dextrose. 37 C.	Dextrose. 20 C.	Lactose. 37 C.	Lactose. 20 C.	Maltose. 37 C.	Maltose. 20 C.	Saccharose. 37 C.	Saccharose. 20 C.	Raffinose. 37 C.	Raffinose. 20 C.	Mannite. 37 C.	Mannite. 20 C.
1	Short	G +	7.3	3.5	.3	0	. .	. .	3.0	.1	1.1	0	0	0
2	Long	G +	3.3	1.9	3.7	0	.9	0	1.8	.1	.2	.1	.1	0
3	Long	G +	3.8	2.4	4.6	.3	1.6	.1	2.6	0	—.1	—.1	0	0
4	Short	G +	6.1	1.8	6.1	.2	2.7	.2	2.1	0	.3	0	0	.2
5	Short	G +	6.5	1.3	2.0	.2	.9	0	1.6	0	0	0	0	0
6	Short	G +	5.0	2.1	5.4	0	1.7	0	.1	1.4	.1	0	.2	.1
7	Short	G +	3.6	1.1	4.1	0	.6	.1	.8	0	.3	0	0	0
8	Long	G +	4.3	1.7	—.1	—	1.5	1.1	1.1	1.3	1.3	0	.2	.2
9	Short	G +	5.7	1.7	—.5	0	. .	. .	1.9	.1	1.1	0	0	0
15	Short	G ∓	4.2	2.6	0	.2	1.6	.4	.1	0	—.1	.2	.1	0
16	Short	G —	.8	.9	.3	.6	. .	. .	0	.2	—.3	0	0	0
17	Short	G —	4.2	2.2	4.3	0	. .	. .	0	0	1.7	0	.1	0
18	Long	G +	3.1	1.8	3.7	0	0	0	0	0	0	0	0	0
19	Long	G +	2.7	1.2	3.4	.5	0	0	0	0	—.1	0	0	0
20	Short	G +	4.5	3.8	. .	.	. .	. .	. .	. .	1.4	. .	. .	. .
21	Short	G +	6.0	3.4	. .	.	. .	. .	. .	. .	1.2	0	. .	. .
22	Short	G +	5.9	3.0	.1	.1	.9	.1	0	0	.1	0	0	0
24	Short	G +	6.0	.5	. .	.	. .	. .	.8	.5	.2	0	. .	. .
25	Short	G +	6.0	1.6	4.1	0	2.9	.2	—.1	0	1.0	0	.1	0
26	Short	G +	6.2	1.4	2.2	0	2.9	.3	0	.2	.1	0	0	0
27	Short	G +	6.3	.3	2.5	0	2.5	.1	2.1	0	.1	0	0	0
28	Short	G +	6.0	.2	3.0	0	2.8	0	1.4	0	—.1	.1	.1	0
29	Short	G +	5.8	2.4	0	0	. .	. .	3.2	0	.9	.1	0	0
32	Short	G +	4.5	0	1.5	0	1.4	0	1.5	.8	1.1	0	0	0
33	Short	G +	5.1	0	.5	0	.5	.2	.9	0	.1	.3	0	0
34	Short	G ∓	2.0	.1	.2	0	.5	0	0	0	.4	0	0	0
35	Short	G +	4.8	.3	2.5	2.2	2.5	2.2	3.5	.1	.2	.1	0	0
36	Short	G +	3.8	.2	.6	.2	.6	0	.1	0	.2	.1	.2	0
38	Long	G +	4.0	.0	1.7	0	1.3	.2	1.1	1.1	0	.1	0	0
43	Short	G —	3.8	.1	3.3	0	0	0	1.5	0	0	0	0	0
44	Short	G +	1.5	0	3.1	0	2.9	0	2.0	.2	.1	0	0	0
45	Short	G —	5.4	0	0	0	. .	. .	.9	0	0	0	0	0
46	Short	G +	. .	. .	. .	.	. .	. .	. .	. .	. .	. .	0	0
47	Short	G +	5.8	0	2.4	0	2.3	.2	2.6	0	.4	0	0	0
48	Long	G +	0	0	.6	.1	.7	.3	0	0	.8	0	0	0
49	Short	G +	4.6	.5	1.2	0	1.5	0	2.4	0	1.1	0	0	0
50	Short	G +	6.0	0	2.6	0	2.5	.2	1.9	0	.1	0	0	0
51	Long	G +	5.4	0	0	0	1.2	0	.2	.1	0	0	0	0
52	Long	G ∓	3.8	—.3	1.6	.1	1.5	0	1.1	.1	.2	0	0	.1
53	Long	G ∓	3.5	0	0	0	2.1	0	.4	0	.6	0	0	0
54	Short	G +	1.1	0	.1	0	.4	0	.8	0	.1	0	0	0
55	Long	G +	1.7	0	.7	0	0	0	.4	0	.1	0	0	0

Chart 1 shows the 20 degree results plotted. Only about 10 per cent. of the throat strains give over 2.5 per cent. acid, while all of the milk cultures give a higher percentage than this.

Lactose: We find here a fairly even distribution of the throat strains at 37 C., while the milk streptococci all give high acidity. At 20 C. lactose proves much more available for the milk than for the throat cultures, as is clearly shown in Chart 2.

TABLE 4.—STREPTOCOCCI ISOLATED FROM "ABNORMAL" THROATS

			Percentage of Acid Formed in											
			Dextrose.		Lactose.		Maltose.		Saccharose.		Raffinose.		Mannite.	
Sample No.	Long or Short Chain Form	Reaction	37 C.	20 C.	37 C.	20 C.	37 C.	20 C.	37 C.	20 C.	37 C.	20 C.	37 C.	20 C.
68	Long	G +	5.5	1.2	0	0	1.8	.4	1.9	1.3	1.4	0	0	0
69	Long	G +	6.2	0	4.7	0	. .	. .	2.1	1.7	.2	0	0	0
30	Short	G +	5.2	2.2	.3	.3	2.0	1.4	2.0	1.8	.1	.2	.1	0
31	Long	G +	2.3	.6	.1	0 0	. .	. .	0	.1	0	0	0	0
37	Short	G +	4.3	1.7	. .	. .	. .	. .	. .	. .	0	.1	0	0
57	Long	G —	3.3	1.9	2.7	2.4	1.7	.8	0	0	.2	0	.3	0
58	Long	G ∓	1.5	.1	1.4	.1	1.8	.8	.9	.1	.4	.1	.2	0
59	Long	G ∓	1.5	.3	1.5	0	.8	0	2.0	0	.3	0	.2	0
60	Short	G +	3.1	0	2.9	0	2.8	0	2.8	0	0	0	.1	0
61	Short	G ∓	1.8	.1	1.8	0	1.9	.2	1.7	0	1.5	0	0	0
62	Short	G +	3.2	0	3.5	0	2.8	0	3.1	0	0	0	0	0
63	Long	G +	1.6	.1	1.6	.1	1.7	0	1.0	0	.3	0	0	0
64	Short	G +	1.9	.3	1.9	0	2.1	.9	.2	0	1.7	.1	.2	0
65	Short	G +	3.4	0	3.2	0	2.8	.8	0	0	1.5	0	0	0
66	Long	G +	. .	. .	1.5	0	1.7	0	.1	0	.3	0	.1	0

TABLE 5.—STREPTOCOCCI ISOLATED FROM MILK

			Percentage of Acid Formed in											
			Dextrose.		Lactose.		Maltose.		Saccharose.		Raffinose.		Mannite.	
Sample No.	Long or Short Chain Form	Reaction	37 C.	20 C.	37 C.	20 C.	37 C.	20 C.	37 C.	20 C.	37 C.	20 C.	37 C.	20 C.
10	Long	G +	4.1	3.4	4.0	0	1.6	0.2	2.0	1.1	1.6	0	.4	0
11	Long	G +	4.1	4.0	.4	0	2.0	1.0	1.0	.4	.2	0	0	0
12	Long	G +	4.4	3.9	4.1	1.5	2.3	1.1	2.3	2.1	.1	0	.1	0
13	Long	G +	3.2	3.9	4.2	4.2	1.3	.5	2.1	2.1	0	.2	.1	0
14	Long	G +	4.3	3.8	4.5	.3	2.1	1.1	1.8	2.0	0	.2	.1	0
23	Long	G +	4.0	5.8	2.2	2.4	1.9	1.9	0	0	—.1	0	0	0
39	Long	G +	5.4	4.8	2.6	2.6	1.6	1.9	0	0	0	.1	0	0
40	Long	G ∓	5.2	4.1	2.5	2.5	1.7	1.4	.1	.1	0	0	0	0
41	Long	G +	4.5	2.8	2.3	1.2	2.0	1.0	.7	2.0	0	.2	0	0
42	Long	G +	4.9	2.9	2.4	.3	2.2	.8	1.6	1.9	.1	.1	0	0
56	Long	G —	3.2	2.4	2.5	2.2	1.5	0	0	.1	0	0	0	0
67	Short	G +	1.2	1.2	2.6	2.6	.7	1.7	. .	. .	.1	.1	0	0
70	Long	G —	4.6	1.2	4.5	.4	2.3	1.2	2.1	.3	0	0	0	0
71	Long	G ∓	4.5	4.0	4.2	.5	2.0	1.0	2.0	2.0	0	0	0	0
72	Long	G ∓	4.2	3.5	4.3	.5	2.4	1.0	2.1	2.0	.1	.2	0	0
73	Long	G —	4.8	3.6	4.4	0	2.2	1.3	2.2	1.9	.1	.1	0	0

Maltose: The throat cultures show a distinct reaction mode, falling between 1.0 and 1.5 per cent. acid. As in the case of lactose, the streptococci from milk are most adaptable as regards their temperature relations.

Saccharose: In this sugar the milk cultures readily produce 2 per cent. acid at 20 C., while the throat strains seldom give more than 0.5 per cent. Reference to Chart 3 shows strikingly this distinction. No other features worthy of particular note are brought out by the 37 degree reactions.

Raffinose: This trisaccharid was rarely fermented and when a reaction was present it was very feeble as shown by reference to the tables.

Mannite: Only a single culture was capable of producing as much as 0.4 per cent. acid with the alcohol mannite. It is obviously of little diagnostic value with throat or milk streptococci.

The results from the cultures isolated from "abnormal" throats showed no essential differences from the normal throat streptococci.

No clue to the relationship of the forms studied was obtained from either morphologic or staining characteristics.

CONCLUSIONS

Our work leads us to make the following tentative conclusions:

1. Streptococci from the human throat and from fresh milk very generally ferment one or more of the sugars, dextrose, lactose, maltose and saccharose, attacking them most readily in the order named. They do not generally ferment raffinose or mannite.

2. The streptococci of the sore throat and the normal throat show no cultural differentiation in relation to the carbohydrates used. Virulence tests might have separated the two groups.

3. The throat streptococci do not readily ferment at 20 C. any of the sugars used, while the milk organisms attack the same sugars and to the same extent at this temperature as at 37 C. This is, perhaps, the most valuable differential feature between chained cocci from these two sources.

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4. Winslow and Rogers: Relationships of the Coccaceæ, 1910.
5. Winslow and Palmer: Jour. Inf. Dis., 1910, vii, No. 1.
6. Gordon, M. H.: Fortieth An. Rep. Loc. Gov. Board, 1910-1911.
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8. Winslow: Ibid.
9. Howe, E. C.: Ibid.
10. Stowell and Hilliard: Ibid.
11. Jackson, D. D.: Jour. Am. Pub. Health Assn., 1912, i, No. 12.

THE EFFECT OF COLD FRESH AIR ON THE BLOOD-PRESSURE IN PNEUMONIA OF CHILDREN *

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It is the general impression of those who have treated children with pneumonia out of doors, or in cold rooms with good ventilation, that, in addition to the increased quiet of the patients and their greater tendency to sleep, the circulatory system is not so profoundly affected and stimulation is less frequently required. Any positive evidence in regard to these impressions has been, however, so far as we are aware, lacking.

In a study of the effects of various circulatory stimulants it was found that a child of 5 years with a febrile pneumonia who received a subcutaneous injection of 1 grain of camphor immediately after being brought in from a balcony in winter, suffered no rise in blood-pressure such as would be expected from the camphor, but on the contrary a decided fall and this fall continued for an hour. As a result of this experience it was decided to test the effect of cold fresh air on the blood-pressure of children with pneumonia and of those convalescing from the disease.

The determination of the blood-pressure of children under 2 years of age by any method involving palpation of the radial artery is so unsatisfactory that the results are usually almost worthless. The children are likely to be so much disturbed as to make the value of any reading uncertain, the arteries are difficult to palpate especially if the pressure is low and the usual cuff is altogether too large. Our results were obtained with a Faught sphygmomanometer which gives very accurate but, we believe, somewhat higher readings than other instruments of the same general type. It allows the point of obliteration of the radial pulse to be determined with great accuracy. This was found to be more reliable than the point of return of the pulse. The reading was always repeated. A special cuff narrower than those usually employed was used. This was rendered necessary by the size of many of the children. The personal element always enters somewhat into the use of instruments of the Riva-Rocci type but with the method which we have employed the variations due to different observers were very slight; in all cases, however, the pressures of each child were read by the same person. No determinations were made unless the child was absolutely quiet and unaffected by the manipulation. The influence of excitement and muscular exertion

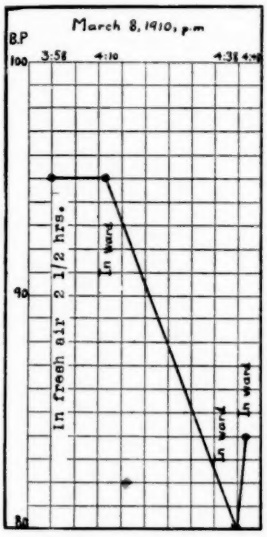
*From the Children's Service of the First Medical Division, Bellevue Hospital, New York.

may, therefore, be eliminated. The readings reported are those of the systolic pressure.

There is even more uncertainty as to the average blood-pressure of normal children at the various ages than as to the average pressure of normal adults, and for very obvious reasons. Taking the observations of Wolfensohn-Kriss,[1] Kaupe,[2] Beretta[3] and Oppenheimer and Bauchwitz,[4] we may consider that the averages are fairly well represented by the following figures:

Age, Years	Blood-Pressure, mm.	Age, Years	Blood-Pressure, mm.
1/12-1	75- 80	5-8	90- 95
1-3	80- 85	8-10	95-100
3-5	85- 90	10-12	100-105

PNEUMONIA



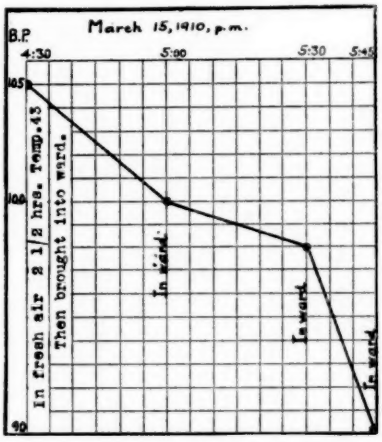


Chart 1.—F. K., aged 8 years. Severe lobar pneumonia and beginning empyema. Operation two weeks later. Recovery. Each square represents five minutes.

Chart 2.—A. M., aged 25 months. Mild bronchopneumonia. Temperature never above 102.5 F. (39.2 C.). Recovery. Each square represents five minutes.

As already stated, we believe that the Faught instrument in the hands of those accustomed to its use gives readings as a rule from 5 to 10 mm. higher than most instruments of the Riva-Rocci type and that averages with this instrument would probably be a little higher than the figures

1. Wolfensohn-Kriss: Archiv. f. Kinderh., 1910, liii, 332.
2. Kaupe: Monatschr. f. Kinderh., 1910, 257.
3. Beretta: Quoted by Oppenheimer.
4. Oppenheimer and Bauchwitz: Arch. f. Kinderh., 1905, xlii, 415.

given. There are, however, marked exceptions to these averages, chiefly on the high side, and it is not unusual to find the blood-pressure of a child 10 or 15 mm. of mercury higher than that of other children of the same age and development. These factors, however, do not influence the value of the variations which are brought about by the influence of cold fresh air, as it is the relative and not the absolute pressure that is of interest.

Studies of the blood-pressure in pneumonia have not allowed the formulation of any definite rules in regard to its behavior. The great

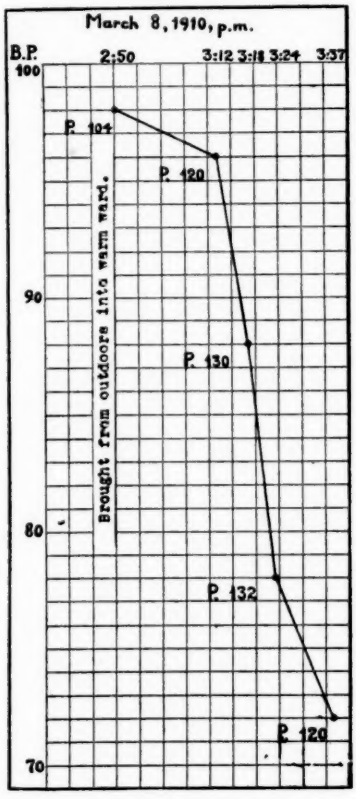


Chart 3.—F. F., aged 5 years. Prolonged extensive bronchopneumonia. Died March 25. Each square represents five minutes.

majority of observations have been made on adults, and irregularly, only one or two in the course of a day, with a great variety of instruments, and very seldom have control readings been made when convalescence has been completed. It is believed by Cook and Briggs,[5] Trumpp[6] and

5. Cook and Briggs: Johns Hopkins Hospital Reports: 1903, xi, 451.
6. Trumpp: Jahrb. f. Kinderh., 1906, lxiii, 43.

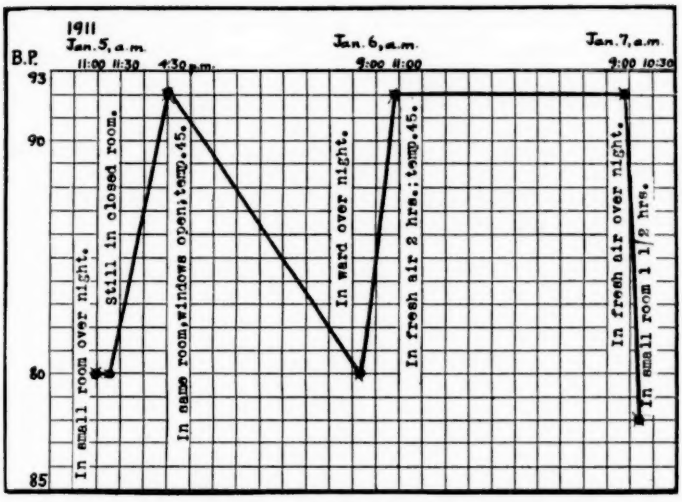


Chart 4.—M. B., aged 2 years. Prolonged severe bronchopneumonia. In hospital from Nov. 10, 1910, to Jan. 10, 1911. Taken home against advice. Each square represents two hours.

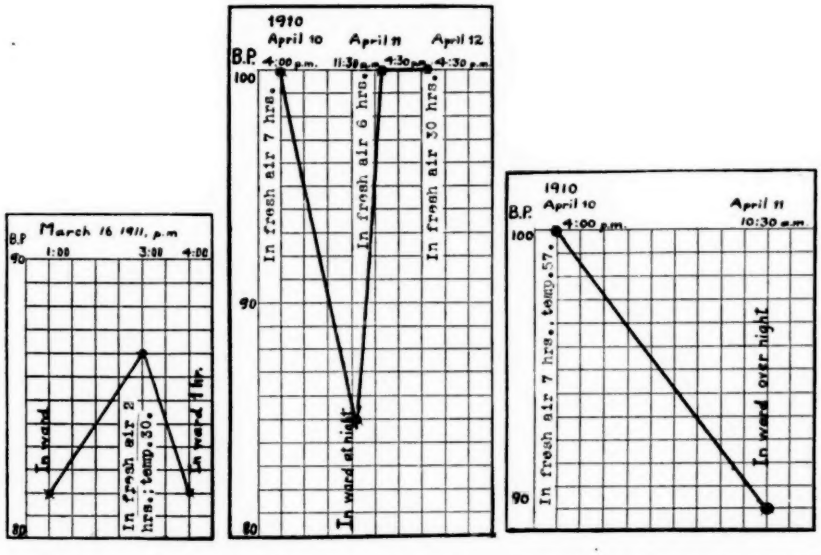


Chart 5.—R. F., aged 6 years. Moderately severe lobar pneumonia, defervescing by lysis on March 18. Each square represents one-half hour.

Chart 6.—T. P., aged 2¼ years. Sharp attack of bronchopneumonia. Temperature normal on April 12. Each square represents six hours.

Chart 7.—C. M., aged 4 years. Bronchopneumonia with very irregular temperature. Defervescence by lysis on April 10. Each square represents two hours.

Muggia[7] that there is an increase in the blood-pressure, especially of children, unless the patients are in collapse. Kaufmann and de Bary,[8] Rongier,[9] Carter,[10] Durand-Viel,[11] Ambard and Beaujour,[12] Ekgren,[13] Külbs[14] and Gilbert and Castaigne[15] believe that the pressure is in general lowered and that it may diminish markedly and suddenly at the time of crisis. Our observations were not made with the object of testing this point, but it is apparent that children seriously ill as were Patients 1, 3, 5, 8 and 10, may have, at ordinary room temperatures, a blood-pressure somewhat below what might be expected at that age. This point can only be determined by a great number of readings carried out throughout the course of the disease.

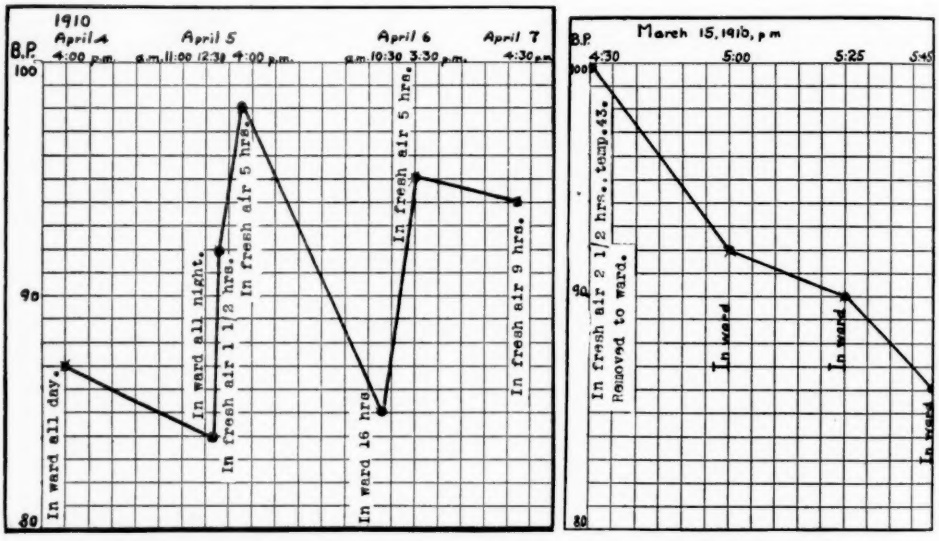


Chart 8.—S. B., aged 8 years. Frank lobar pneumonia. Defervescence by crisis on April 6. Each square represents three hours.

Chart 9.—M. L., aged 2 years. Bronchopneumonia. Defervescence by crisis March 16. Each square represents five minutes.

Our patients were treated in the wards of Bellevue Hospital, which are thoroughly ventilated and usually kept at a temperature of about 65 F. (18 C.), or on the balconies outside of the wards. In the wards

7. Muggia: Quoted by Beretta.
8. Kaufmann and de Bary: Berliner klin. Wchnschr., 1888, xxv, 557.
9. Rongier: Thèse de Paris, 1900.
10. Carter: Am. Jour. Med. Sc., 1901, cxxii, ii, 854.
11. Durand-Viel: Thèse de Paris, 1903.
12. Ambard and Beaujour: Arch. gén. de méd., 1904, i, 520.
13. Ekgren: Zeitschr. f. klin. Med., 1905, lvii, 411.
14. Külbs: Deutsch. Arch. f. klin. Med., 1907, lxxxix, 457.
15. Gilbert and Castaigne: Rév. gén. de clin. et de pract., 1899, xiii, 769.

they were covered lightly with blankets; outside they were clothed in woolen wrappers and covered with many blankets, according to the temperature. Hot water bottles were at their feet and they wore hoods. Only the faces were exposed. None was receiving circulatory stimulation at the time that the readings were made. The accompanying charts show some of the results on the blood-pressure in children divided into two classes—the heading "pneumonia" indicating that they were still suffering from the disease and were in the febrile stage, and "convalescent" that they had defervesced, although not necessarily free from pulmonary signs.

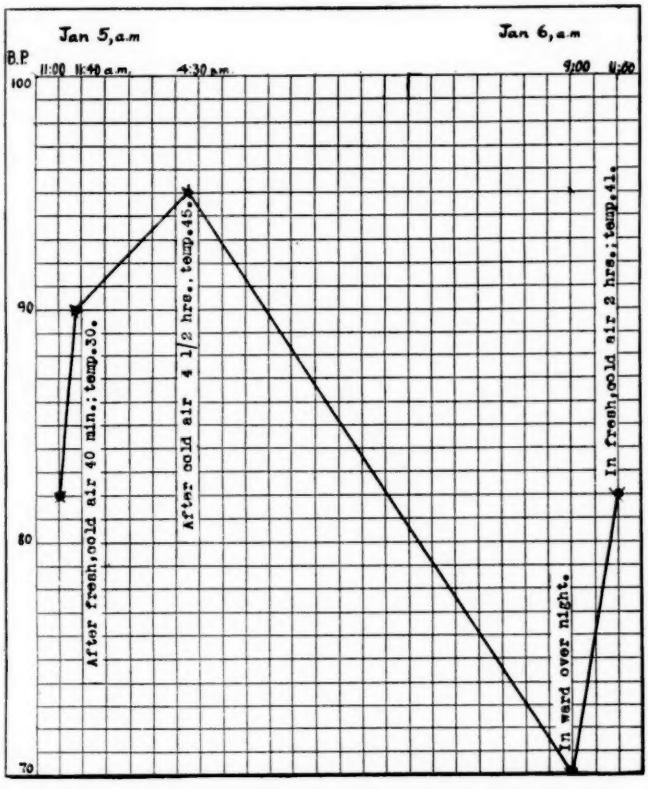


Chart 10.—H. G., aged 3 years. Lobar pneumonia running a severe course. Temperature normal on January 7. Each square represents one hour.

It will be seen from an examination of the charts that the effect of cold fresh air in patients with active pneumonia was always to produce a rise in blood-pressure and that removal to a warm but well-ventilated ward was to produce a fall in blood-pressure. We have never found this to fail, although in one or two instances the variation in pressure has not been as noticeable as in most of the charts shown. The rise is not apparent until half an hour or more, sometimes not until an hour, after

being put out of doors, and it does not reach its maximum for about two hours. Thereafter the effect, so far as we have observed, is continuous for even as long as thirty hours and we have not observed any tendency for the pressure to fall as if from exhaustion of the effect. We have not made observations on any fatal cases that were treated continuously out of doors and are therefore unable to say what would be the behavior of the blood-pressure in cases growing constantly worse and progressing to a fatal termination.

CONVALESCENT

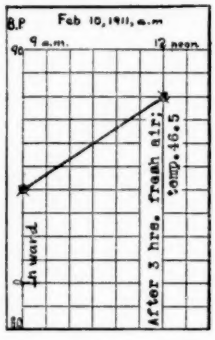


Chart 11.—M. B., aged 18 months. Two weeks after pneumonia. Each square represents one-half hour.

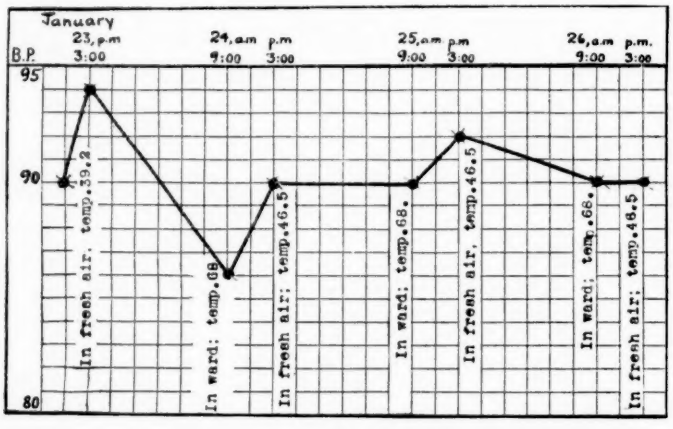


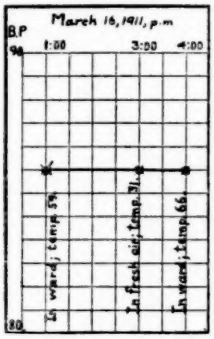
Chart 12.—C. H., aged 10 years. Moderately severe lobar pneumonia. Temperature normal for ten days. Each square represents three hours.

On the other hand, after removal from the cold fresh air the blood-pressure falls rapidly; the fall is apparent in fifteen or twenty minutes and usually reaches its lowest point in an hour, to remain at this minimum unless influenced by the course of the disease, by stimulation, or by the child's again being placed out of doors, when it again rises.

In convalescents, on the contrary, the results are usually much less striking and may even be absent. Instead of a rise of 10 or 15 mm. of mercury, the difference is only a few millimeters or none at all.

Chart 15 is an exception and shows as great an effect as is found during the acute febrile period. The pressure in the ward was 80 mm., or about 10 mm. less than would be expected in health. Whether this low pressure, which is occasionally found in convalescence, as is mentioned by Weigert[16] and other authors, can be thus influenced, in contradistinction to the pressure which is at or near normal, must remain undecided until a far greater number of observations can be made. We believe that it is very likely that this is the case.

The number of our observations have not been sufficiently numerous to allow us to say whether those children with a very low blood-pressure respond better than those whose pressure is nearly normal.



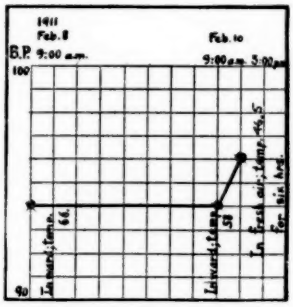


Chart 13.—D. W., aged 18 months. Had defervesced on March 15 after a mild attack of bronchopneumonia.

Chart 14.—B. A., aged 9 years. Mild lobar pneumonia. Defervescence by lysis. Temperature normal February 7. Each square represents six hours.

We have been unable to produce results by putting children out of doors in warm weather; as the temperature increases in the spring and the out-of-doors temperature approaches that of the ward, the effects are less and less marked until none whatever is produced. It would seem, therefore, that the all important factor is the cold.

The influence on the blood-pressure is undoubtedly brought about by reflex stimulation of the vasomotor center by the action of cold air on the skin of the face and on the nasal mucous membrane. No other part of the child is exposed and there is no additional factor that can be supposed to play a part. It is striking that this reflex should produce such a constant and continuous effect. As has been shown, the pressure remains at a higher level for hours, shows no tendency to fall and even

16. Weigert: Samml. klin. Vortr., 1907, Innere medizin, 138.

after a return to the ward does not fall to a point below that previously obtaining at the ward temperature.

As to the beneficial effect of cold air on the circulation, there can be no doubt that when the pressure is abnormally low an increase in this, which is accomplished without disturbance or irritation, which is constant and continuous and which causes no exhaustion or bad effects, is of the greatest value. We are quite aware that the determination of the blood-pressure alone does not give all the data in regard to the circulation, such as the mass movement of blood, etc., but taken with the ordinary methods of physical examination it is the best indication of the condition of the circulation that we possess. Chart 3 raises the question whether the cold by increasing the pressure to 98 mm. lessened the demands on the heart,

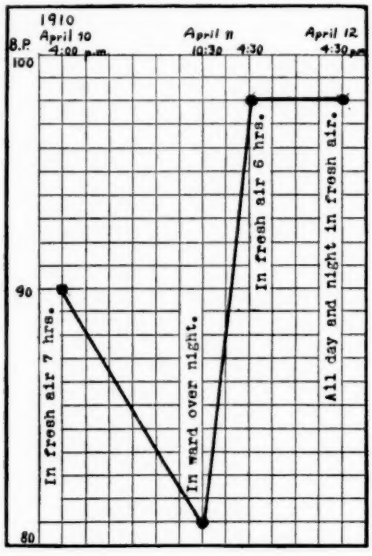


Chart 15.—A. S., aged 5 years. Lobar pneumonia, moderately severe. Temperature normal April 9. Each square represents three hours.

as indicated by a pulse-rate of 104, and whether the withdrawal of cold with the consequent fall of pressure to 72 demanded an increase of cardiac exertion, as indicated by a rise of the pulse to from 120 to 132. This leaves entirely out of account the other beneficial effects of cold fresh air.

The symptoms of the death of children from pneumonia are those of vasomotor failure. It is rare to see intense cyanosis, edema of the lungs and evidences of right heart incompetency. Whether under such circumstances cold air would have an influence in assisting the circulation we are unable to say.

Whether it is advantageous from the standpoint of the circulation to raise a blood-pressure which is at an average point or slightly above this is a question which we cannot at this time answer. It may be that the pressure is only maintained at this point by an unnecessary cardiac exertion accompanied by a very rapid pulse and that a high pressure, reflexly produced through the vasomotor system, may allow of greater rest to the heart. We are confident that we have observed no bad effects on the circulation nor have any been reported, notwithstanding the prevalence of the cold air treatment of pneumonia and the lack of selection of patients to be treated by this method. It is possible that from the beginning of the disease when the blood-pressure is not low, cold air may have a beneficial tonic action on the vasomotor center so as to render it less likely to be profoundly influenced by the organisms or their products, and it seems to us very likely that the other beneficial effects of cold air may so modify the disease as to render less likely the development of partial or complete circulatory failure.

A STUDY OF ICTERUS NEONATORUM BY MEANS OF THE DUODENAL CATHETER *

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The occurrence of jaundice within the first few days of life has, as might be expected, stimulated repeated investigation. The theories advanced to account for this striking phenomenon have been most ingenious, but being based quite as much on speculation as on fact, they have proved unsatisfactory and have been accepted only tentatively in lieu of something sounder. The excuse offered for entering on a path so well trodden is that a new point of vantage of this subject now presents itself, through the use of the duodenal catheter, recently described in quite another connection in the pages of this journal.[1] By this means direct access to the bile is afforded at its site of excretion. Before presenting the data gathered by this means, however, it would seem of advantage to sketch in outline the present status of this question.

It is to be noted at the outset that there is a surprising lack of unanimity even as to the incidence of icterus neonatorum. This is all the more remarkable when we consider that this phenomenon is of so simple a nature that it should not allow of a wide variance of opinion. Nevertheless we find the incidence given by some as 15 per cent., by others as 85 per cent., and by still others placed at various levels between these extremes. This marked divergence must be attributed almost entirely to differences in method of examination. It would seem that those who made a point of examining with the aid of strong daylight, and of looking for the faint yellow tinge on the back or on the nostrils, rather than in the sclera, as in the case of adults, have observed icterus in at least 75 per cent. of infants. In a series of 100 infants whom I examined for this sign, it was definitely noted eighty-three times. But even this figure probably does not give us a correct idea of its frequency, for it is manifestly impossible for us to detect by means of mere clinical inspection the presence of a trace of bile in the blood or in the skin, and when we consider the crudeness of the test, and remember that bilirubin crystals frequently have been found in the tissues of infants who showed no jaundice during life, [2, 3] we probably do not err in believing that there are few instances in which bile does not enter the general circulation

*From the Research Laboratory, Board of Health, New York City.

1. Hess, A. F.: Am. Jour. Dis. Child., March, 1912.
2. Neumann, E.: Virchows Arch. f. path. Anat., 1888, cxiv, 394.
3. Orth, J.: Virchows Arch. f. path. Anat., 1875, lxiii, 446.

soon after birth. In view of its frequency, icterus neonatorum must therefore be considered a peculiar entity, the sole example of a physiologic icterus.

The opinion that this icterus is hematogenic in origin, that the bile is formed directly from the blood pigment, does not need more than passing mention, since it was proved experimentally by Minkowski and Naunyn[4] and by others that if the liver is extirpated jaundice cannot be induced; and since Birch-Hirschfeld[5] demonstrated bile acids in the pericardial fluid of infants who had manifested icterus neonatorum during life. The blood may likewise play a rôle in the causation of this form of jaundice. The presence of bile acids in the body fluids, however, conclusively showed the participation of the liver.

Although the hematogenic theory is certainly insufficient to account for this form of icterus, or indeed for any icterus whatsoever, it has been accorded a position of varying importance by many writers who believe in the hemohepatogenic origin of this disturbance. Hofmeier[6] was the first to advance the theory that in the new-born there is a marked destruction of red blood-cells in the circulation, that in cases of icterus neonatorum this takes place to an exceptional degree, and that hemoglobin thus set free forms the basis for an excess of bile formation in the liver (polycholic or pleiochromic icterus). Knöpfelmacher[7] has failed to find this destruction of red blood-cells and therefore gives this theory no place in the etiology of this form of icterus. However, very recently[8] blood examinations have again been reported to uphold it. In view of the fact that great weight has been attached to this etiologic factor, we should bear in mind that free hemoglobin has never been found in the new-born in the circulating blood. And what is of far greater moment in controverting the claim that this hemolysis is alone sufficient to account for the icterus is the experimental evidence that jaundice cannot be produced by intravenous injections of hemoglobin, and the clinical experience that it does not supervene following transfusion of blood.

Another theory which cannot be passed by without mention, as it prevailed for many years, was that of Frerichs.[9] The distinguished author of "Diseases of the Liver" explained this jaundice by reasoning that coincident with birth, and closely following the circulatory changes in the umbilical vessels, there results a marked anemia and decrease in pressure in the liver capillaries, which in turn leads to a diminished pressure in the bile capillaries, and an overflow of bile into the blood stream. Quite apart from the fact that this explanation is purely hypothetical, it can

4. Minkowski and Naunyn: Arch. f. exper. Path. u. Pharmacol., 1885, xix, 34.
5. Birch-Hirschfeld: Virchows Arch. f. path. Anat., 1882, lxxxvii, 1.
6. Hofmeier: Ztschr. f. Geburtsh. und Gynäk., 1882, viii, 287.
7. Knöpfelmacher, W.: Wien. klin. Wchnschr., 1896, No. 43.
8. Heimann, F.: Ztschr. f. Geburtsh. und Gynäk., 1912, lxix, 165.
9. Frerichs: Klinik d. Leberkrankh., 1858, i, 198.

no longer be given any weight, in view of the marked congestion of the hepatic vessels, in contradistinction to an anemia which has been uniformly found at autopsy of the new-born.

The theory to-day which, according to Finkelstein,[10] finds the greatest acceptance is that of Quincke.[11] This author believes the patency of the ductus venosus to be the deciding etiologic factor. If this connecting link between the portal and systemic venous circulation fails of obliteration at birth, icterus results, as bile passes directly from the meconium in the intestine to the portal vein, and circumventing the liver, enters the inferior vena cava. There are other subsidiary causes which play a part, such as the polycholia, the small amount of bile pigment excreted by the kidneys, and the peculiar nature of the intestinal contents, the bile containing meconium. As this theory enjoys such wide acceptance, it demands particular consideration, and it is therefore remarkable that although it was promulgated some twenty-seven years ago, so little experimental work has been undertaken to confirm or refute its validity. Quincke himself investigated the condition of the ductus venosus in only five cases, and in these reported on the circumference of the vein rather than on its lumen, which would seem to be the more important factor. Schreiber[12] supported this theory by citing the figures of patency of the ductus venosus published by Elsässer[13] many years ago. However, these figures may well be used against Quincke's hypothesis. In ninety-two autopsies on infants born alive, the ductus venosus was found patent in sixty-five, and in fifty of these where the infant was seven days or more of age, the ductus was patent in thirty-one and closed in nineteen instances. This includes some premature infants, and omits some cases in which post-mortem conditions seem insufficiently described. In the second week of life the ductus was patent in nineteen out of twenty-seven cases, in the third week in eight out of fifteen cases, and also in four of eight dissections made at a still later period of life. It is needless to add that if we use this anatomic study as a criterion, we should not be led to consider this icterus as a consequence of a patent ductus venosus; for if such were the case, icterus neonatorum would be a phenomenon not of the first week but of the first month of life.

It should be remembered that Elsässer carried out this investigation purely for medicolegal purposes. There has been no investigation to correlate anatomic findings of this nature with the clinical occurrence of icterus, and without this link these mere dissections can furnish but inconclusive evidence. It would seem worth while to study the patency

10. Finkelstein, H.: Lehrbuch d. Säuglingskrankh., 1905, i.
11. Quincke, H.: Arch. f. exper. Path. u. Pharmakol., 1885, xix, 34.
12. Schreiber, E.: Berl. klin. Wchnschr., 1895.
13. Elsässer: Ztschr. f. Staatsarzneikunde, 1841, xli.

of the ductus venosus also in animals and to determine whether it bears any relation to jaundice.

Knöpfelmacher[14] probed this theory by attempting to remove the meconium from the bowel shortly after birth. With this end in view he used repeated high enemata, employing acidulated water in order not to dissolve the bilirubin, and performed four to eight irrigations of this nature. Although this method is ingenious, its results do not seem conclusive; for on scanning the notes of the twenty cases on which this experiment was carried out, it is evident that the bowel was not completely emptied of meconium. In most cases a "milk-stool" did not appear until the third or the fourth day of life, which must entirely invalidate the test. Furthermore, only one case is noted in which jaundice made its appearance later than the milk-stool, and on this point the entire experiment would seem to hinge — the occurrence of jaundice after and in spite of the emptying of the intestine of all meconium. The question does not seem to be open to solution by this method, for it is impossible to be certain that meconium is not still present in the bowel. Indeed, in this connection Kehrer's[15] figures showing that the jaundice bears no relation to the date of disappearance of the meconium, and the appearance of "milk-stools," and that among his 123 cases, when the yellow stools appeared late there was no increased frequency or intensity of jaundice, seem quite as convincing as Knöpfelmacher's carefully planned experiment.

There are many other theories which might well be mentioned, but we shall limit ourselves to an outline of a group which may be designated as that of bile stasis. It is unnecessary to consider such hypotheses as an obstruction of the bile-ducts by mucus or epithelium (Virchow), or a congenital narrowness of these ducts (Kehrer), or compression of the larger ducts through edema of Glisson's capsule, as post-mortem examination has failed in every instance to confirm them. There are other investigators, however, who believe that this stasis of bile results from inadequate excretion, that the bile is forced out of the intracellular biliary capillaries into the liver cells and then enters the blood stream. Browitz[16] has supported this theory with careful microscopic examinations which have been confirmed by Knöpfelmacher. Neither believes that the jaundice is due to mechanical obstruction, as no tears in the fine bile ducts are found, merely varicosities, but rather to a disturbance of function of the liver cells allowing a reverse current of bile into the blood stream. In stillborn infants Knöpfelmacher[14] describes an increased viscosity of the bile, but in infants of the first few days of life a markedly decreased viscosity with an abnormal distention of the bile capillaries. The viscosity

14. Knöpfelmacher, W.: Jahrb. f. Kinderh., 1908, lxvii, 36.
15. Kehrer: Oesterr. Jahrb. f. Pädiat., Wien, 1871, p. 71.
16. Brewitz: Wien. klin. Wchnschr., 1900, p. 785.

tests were carried out according to Ostwald's method. However, the bile was obtained from the gall-bladder and not from the hepatic ducts, and may have been a fetal secretion. The histologic investigations showing bile stasis are especially welcome, as they do not burden the already overweighted etiology of icterus neonatorum with another theory.

INVESTIGATION WITH DUODENAL CATHETER

It is possible to pass the duodenal catheter in the new-born infant with as great ease as in infants a few months of age. The technic is the same; however, a 14 (F.) Nélaton catheter is used instead of the larger 15 (F.) catheter. There is a tendency to slight bleeding due to the delicate texture of the mucous membrane during the first hours and days of life, and for this reason aspiration must be gentle.

It seemed that the first point to ascertain should be the time when excretion of bile begins in the new-born. Accordingly the catheter was passed on infants a few hours old; indeed, in four cases within two hours of birth. It is advisable from an etiologic point of view to divide the cases into groups, those tested before they had nursed, and those tested subsequent to nursing. The former group comprises fifty-two cases, almost all under 6 hours old, the time when the infant was usually put to the breast. It includes, however, in addition to four infants under 2 hours old, several 12 hours old. The test was carried out generally in two stages, and the catheter was passed into the duodenum in the manner previously described, and after ten minutes of intermittent aspiration withdrawn; it was then washed out and reintroduced for the same length of time. Dividing the test in this way has the advantage of avoiding an error which might be occasioned by the plugging of the lumen of the catheter with mucus, an event which would of course nullify the test if it occurred soon after the intestine was entered. It is of further advantage, at it happens frequently in the new-born, owing to the pressure of the pyloric or cardiac sphincter, that it is impossible to aspirate viscid fluid, and the bile or intestinal juice is obtained only after the catheter is withdrawn into the stomach or even into the esophagus. For these reasons the two tests furnish a safer criterion. Where any doubt as to the reliability of the test was entertained, aspiration was continued for a longer period.

Bile was obtained but once in the course of these fifty-two tests, and in that instance in small quantity from an infant 2½ hours old (Table 1). It may therefore be stated that in the first half day of life there is rarely an excretion of bile into the intestine. It is possible, had the test been continued for many hours, a procedure which did not seem justifiable, bile might have been oftener encountered. However, the aspirations were sufficiently numerous to warrant the deduction that bile, in general, is not excreted during this early period.

Nineteen tests were made on infants from 12 to 36 hours old. It was found that where marked jaundice was present, in four cases, bile could be obtained, but that where it was absent, in fifteen cases, bile also was absent. Mere statistics, therefore, cannot give a correct idea of the bile excretion of this period, for they will vary according to the number of cases of marked jaundice which they comprise. This is true to some extent in infants 1½ to 3 days old. As the infants increased in age I found bile in increasing frequency; never to the same extent, however, as in the deeply jaundiced infant (Table 1). There is no doubt that in some instances although bile was not obtained, it must have been excreted to some extent into the gut. This was to be expected when we consider that although bile is secreted continuously it is excreted intermittently. In this connection we may refer to Table 2, composed of cases in which numerous tests were carried out on the same infants on consecutive days. A survey of this table shows that when bile was obtained it was almost always found in successive examinations, and that its presence was not subject to caprice.

TABLE 1.—RELATION OF AGE TO EXCRETION OF BILE (DUODENAL CATHETER)

No. of Cases	Age	No Bile	Bile	Marked Jaundice	Remarks
52	½-12 hrs.	51	1	0	39 cases were less than 6 hours old.
19	12-36 hrs.	15	4	4	Marked jaundice in all four positive cases; in others no bile obtained.
15	1½- 3 days	5	10	6	Marked jaundice in four positive cases; in other two jaundiced cases bile next day.
13	3- 4 days	4	9	6	Jaundice not increasing.
12	4- 5 days	2	10	5	Jaundice decreasing.
13	6-11 days	2	11	4	Jaundice decreasing.
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In twenty-four cases a careful study was carried out of the relation of the bile excretion to jaundice. In some of these cases the catheter was employed in a routine manner soon after birth, and then every day subsequently until bile was obtained on two successive occasions. These infants were unselected. In eleven of the cases, however, a selection was made, based on the occurrence of early or marked jaundice. Seventy-two tests in all were carried out, the results of which are given in Table 2 (a) (b). A most striking and consistent fact was that bile was not obtained in any case previous to the appearance of jaundice. Even where bile was obtained within the first twenty-four hours, a marked early jaundice also manifested itself (Cases 9 and 19). It is evident that the quantity of bile secreted plays an important rôle. In one case (Case 3), for example, excretion was delayed, and bile was not obtained until the seventh test, so that unless secretion of bile had been exceptionally meager, this certainly must have resulted in marked icterus. On the contrary, a slight ephemeral jaundice was noted on the second day. And again if we

TABLE 2.—RELATION OF JAUNDICE TO BILE EXCRETION (DUODENAL CATHETER)

(*A*) *Unselected Cases*

Case	Age, Days	Bile*	Jaundice	Food Supply	Remarks
1	5/8	—	0	Colostrum +	
	2⅔	+	+	.....	
	3⅔	0	++	.....	
	4⅔	0	—	.....	
2	⅛	—	—	Colostrum +	
	1⅛	—	+	Colostrum +	
	2⅛	—	++	Colostrum ++	
	3⅛	+	++	Milk +	Ten-minute test.
	4⅛	+	++	.....	Ten-minute test.
3	¼	—	—	Not fed.	
	1¼	—	+	Colostrum +	
	2¼	—	—	Colostrum ++	
	3¼	—	—	Colostrum ++	
	4¼	—	—	Milk.	
	5¼	—	—	.....	Bile in stool
	6¼	+	—	.....	Ten-minute test.
	7¼	—	—	.....	Castor-oil given shortly before test.
	8¼	+	—	.....	Five-minute test.
4	¼	—	—	Not fed.	
	1¼	—	—	Colostrum —	Ten-minute test.
	2¼	+	—	Milk.	
5	⅛	—	—	Not fed.	
	1⅛	—	+	Colostrum +	
	2⅛	—	++	Colostrum ++	
	3⅛	+	++	Milk.	Bile in second test.
	4⅛	+	++	.....	Bile in both tests.
	6⅛	0	++	.....	
6	1	—	+	.....	Supplementary feedings begun at one hour.
	2	—	—	.....	
	3	—	—	.....	
	4	+	—	.....	
7	¼	—	+?	Not fed.	
	1¼	—	+	Colostrum +	
	2¼	+	+	Milk.	Bile in both tests.
	3¼	+	++	.....	Bile obtained at once.
8	⅛	—	—	.....	
	1⅛	—	+	.....	
	2⅛	+	±	.....	
9	1	+	+	.....	Bile in second test.
	2	++	++	.....	Bile in first and second test.
10	½	—	—	.....	Weight 6 lbs. 13 oz.
	1½	—	+	.....	Weight 6 lbs. 10 oz.
	3½	—	+++	.....	Weight 6 lbs. 9 oz.
	4½	—	+++	.....	Weight 6 lbs. 10 oz.
	5½	+	+++	.....	Weight 6 lbs. 11½ oz. Ten-minute test.
	6½	+	++	.....	Weight 6 lbs. 13½ oz. Ten-minute test.
11	11/12	—	+	.....	**Early jaundice,**
12	1⅓	—	—	.....	Supplementary feeding begun at two hours.

TABLE 2.—*Continued.*

Case	Age, Days	Bile*	Jaundice	Food Supply	Remarks
	3⅓	+	++	.....	Bile in stomach.
	4⅓	+	+	.....	Ten-minute test.
13	1¼	—	+	.....	
	2¼	—	0	.....	
	3¼	—	—	.....	
	4¼	+	—	.....	Bile obtained at once.
	5¼	—	—	.....	
		(B) Cases Selected on Account of Early or Marked Jaundice			
14	3¾	+++	+++	.....	Bile in stomach.
	4¾	0	++	.....	Bile in stomach.
15	4½	+++	+++	.....	Bile in stomach. Two-minute test.
	5½	..	++	.....	Bile in stomach. Two-minute test.
16	2	+++	+++	.....	A case of "Family Icterus." Infant saffron colored, skin dry, marked loss of weight, anuria. Bile in stomach at each test.
	3	+++	+++	.....	
	4	++	+++	.....	
	5	++	+++	.....	
	6	+	++	.....	
17	6	+	+++	.....	Jaundice decreasing.
18	3½	+	++	.....	
	4½	+	++	.....	Jaundice decreasing.
19	5/6	+	++	.....	
20	3½	++	++	.....	Bile in stomach and vomitus.
21	1½	++	++	.....	Bile in stomach and vomitus.
22	3	++	++	.....	Bile in stomach.
	4	++	+++	.....	Bile in stomach.
	5	++	++	.....	
23	6¾	+	+++	.....	Bile in stomach.
24	8½	+	+++	.....	Jaundice decreasing.

*+=Present; —=absent; 0=no examination.

compare a case in which there was no jaundice (Case 4), with another in which there was the most intense saffron jaundice, we find that in the latter instance the excretion of bile began earlier and was incomparably more profuse; indeed, in the jaundiced case the excretion of bile seemed almost continuous. Had the jaundice depended solely on the excretory function of the liver, the amount of bile secreted being the same, conditions must have been reversed. The importance of the secretion in this connection is further emphasized when we compare the columns of the table and note the general parallelism of marked jaundice and marked bile. And yet the efficiency of excretion also plays a part, for it is clear that if excretion were adequate there would be no stagnation of bile, and the dilatation of the biliary capillaries, which is a definite and constant histologic accompaniment of icterus neonatorum, would not occur. It has, furthermore, been repeatedly shown by animal experimentation that

jaundice cannot be effected, however greatly we may increase the secretion of bile, unless excretion is at the same time hampered. However, experiments of this nature on normal new-born animals would be of exceptional interest.

There are some other points noted in this table which require a word of explanation. In many of the cases a record was made of the time when colostrum appeared in the mother's breasts, of its profuseness, and of the day on which milk was first secreted. This was done with the purpose of ascertaining whether these facts bore any relationship to the occurrence of jaundice. In addition, a number of infants, some of whom are included in this table, were given colostrum within the first few hours of life, expressed from the breasts of mothers whose children were 2 to 3 days old. The object of these tests was to determine whether, as has been claimed, starvation of the infant during the first day or two of life is an etiologic factor of the icterus. It was soon evident that it was a consideration of little moment; in the case showing no jaundice, the colostrum was noticeably scanty (Case 4), whereas in an instance in which colostrum feeding was begun when the baby was 2 hours old, jaundice was marked (Case 12). Furthermore, jaundice developed in a baby which gained 7 ounces during the first four days, owing to the fact that the mother's milk was profuse almost immediately after delivery. These incidents also show that the occurrence of jaundice but once in the series of fifty-two infants who had not been fed, was due not to the lack of food but to their time of life. The weights of a number of infants have been recorded, as a loss has been emphasized by many writers. It is not exceptional, however, for an infant with marked jaundice to lose but a few ounces and to regain its birth weight within the first week (Case 4). We have not considered critically the bearing of prematurity, of asphyxia, and of breach presentations in the etiology. It has been interesting as well as instructive to note how the predominance of jaundice in these abnormal conditions has been brought forward to strengthen the most divergent theories, from that of Morgagni, who laid the burden on the diminished pressure in the hepatic circulation, to those of the present day.

SUMMARY

Tests by means of the duodenal catheter show that bile is very rarely excreted during the first twelve hours of life; it was obtained but once in the course of fifty-two tests.

Bile excretion during the subsequent twenty-four hours is variable; in cases of marked jaundice it is profuse; in cases not jaundiced it is scanty or absent.

The function of excretion gradually becomes fully established during the first week or ten days of life.

Where jaundice manifests itself, it precedes the excretion of bile into the duodenum.

Secretion of bile varies within wide limits. In general it is marked when the jaundice is marked.

The occurrence of jaundice results from a defective correlation of excretion and secretion. It is generally caused by the inability of the rudimentary excretion to cope with the sudden profuse secretion of bile.

CONCLUSIONS

Icterus neonatorum has always been the favorite domain of hypotheses, and happily I have no new theory to introduce into this overcrowded realm. It would seem beyond doubt that in most cases there is a disturbance of secretion, a polycholia, or according to Stadelman, a mere increase in the formation of bile pigment (pleiochylia). In many of the cases of marked jaundice this hypersecretion reaches an intensity so marked that bile overflows into the stomach, where it may be found on introducing the stomach tube, or manifests itself by its presence in the vomitus. The cause of this pleiochylia is not at present definitely proved, so that it would seem advisable in this instance to follow the principles of the physiology of bile secretion and assume, what seems probable, that it is due to an increased amount of available hemoglobin; that bile salts are absorbed from the intestine, resulting in a disintegration of blood-cells and a consequent increased elaboration of bile in the liver. Whether or not this explanation of its histogenesis prove correct does not alter the fact of the usual occurrence of a hypersecretion.

In addition to the disturbance of secretion there is a coincident disturbance of excretion, and it is probable that were excretion as well provided for at birth as in later infancy, there would be no disturbance such as icterus neonatorum. We find, however, that the liver is most inadequately equipped to excrete this excess of bile; that during the first hours of life there is practically no functionating excretory mechanism, and that this function manifests itself but gradually. The bile is secreted under low pressure, but where an excess is secreted, as is generally the case in jaundice, the pressure is proportionately increased, so that a profuse excretion of bile into the duodenum is obtained. Nevertheless, congestion of the biliary capillaries ensues, as is shown by histologic investigations, and icterus results.

There has been too little consideration of the interrelation of secretion and excretion. The former may be said to be the more important, for in some cases in which there is but slight secretion, the rudimentary excretory function suffices to prevent the manifestation of icterus. That the lack of excretion cannot be the sole cause of the disturbance is evident when we consider that several cases of congenital obliteration of the bile

ducts have been reported[17] in which icterus did not develop for days or even weeks, and yet this lesion may be considered comparable to the complete tying off of the common bile duct at birth. The complementary parts which secretion and excretion play in icterus neonatorum may well be compared to these functions in the breast after parturition. If the excretion of milk is copious and adequate, no pathologic phenomena result; however, if it prove inadequate, stasis ensues, lactose enters the blood stream and appears in the urine. The lactose is the counterpart of the bile. The reason why jaundice appears in the first days of life is because at the time when excretion has incompletely assumed its function throughout the body, in the liver as well as in other organs, for example, in the breasts and in the kidneys, a sudden flood of bile is poured into the passive excretory ducts and gains access to the hepatic circulation.

154 West Seventy-Second Street.

17. Thomson, J.: Congenital Obliteration of the Bile Ducts, 1902.

COMMUNICATION CONCERNING EIWEISSMILCH

To the Editor:—In the November number of your journal in an article entitled "The Use of Malt Sugar and High Percentages of Casein in Infant Feeding," Dr. Morse claims to have treated cases of nutritional disturbance in infants with a mixture based on the principles of eiweissmilch, and is not very well satisfied with the results. We, however, are still enthusiastic about eiweissmilch, having seen its success in hundreds of cases, and believe Dr. Morse's conclusions may be due to an improper understanding of the technic and principles of feeding.

For example, on page 318, he says we advise giving 180 to 200 calories per kilo of body weight, and on page 326 that it is difficult to get children to take this amount. This is, of course, a gross error. We recommended not 180 to 200 calories, but 180 to 200 c.c. of eiweissmilch which, without addition of sugar, means only 70 calories per kilo.

In another place Dr. Morse says that we "seem to imply that all disturbances of nutrition occur independently of bacterial action." We do not remember ever taking this standpoint. Many publications from our clinic explain that there *must first* be an *injury* of the intestinal epithelium caused by *abnormal bacterial fermentation,* before the food can itself be harmful.

On page 320 Dr. Morse says that among other reasons, we recommend the addition of buttermilk to our mixture on account of its low salt-content. This is a misunderstanding. We use buttermilk because it is a fat-free milk, with a decreased quantity of lactose.

Now a word about Dr. Morse's results. He does not like and use eiweissmilch for a routine food, but being prepared to take advantage of the main principles of the method he employs another mixture, prepared with precipitated casein from one quart of milk, and cream containing a high percentage of fat with addition of dextrin-maltose. He does not say how much cream there was in the mixture, but from the fact that it was extremely high in fat, the quantity must have been low and we can rather calculate it. His mixture contained 2 per cent. protein, 1.8 per cent. of which was precipitated casein. This leaves only 0.2 per cent. protein from the cream as is found in about 60 gm. The content of whey is not mentioned. From this we cannot think that whey was added. Probably the mixture contained only the little quantity of whey that was found in the cream.

We believe that this very low content of whey, and therefore of salts, explains why Dr. Morse's children did not gain. A child needs not only sufficient calories of protein, fat and carbohydrate, but he needs also a *definite minimum* of salts contained in *the whey.* Give a child too little whey and he rapidly develops a salt hunger, the first symptom of which in strong children is a cessation of the gain in weight and in weak children, with nutritional disturbances, is a rapid fall in weight, even threatening death, though the caloric value of the food may be normal.

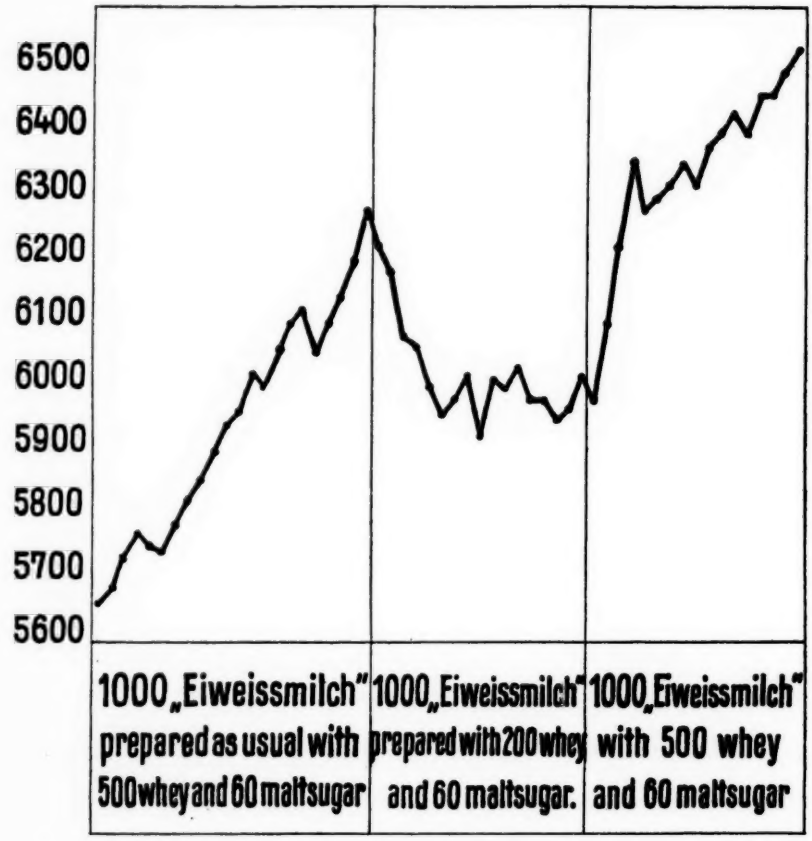

Chart showing loss of weight in grams in a healthy child on too great reduction of the quantity of whey, protein, fat and carbohydrate being sufficient.

We show, as illustration, a curve of a healthy child who got sufficient calories of protein, fat and carbohydrate, but who promptly got a loss of weight and did not thrive when the whey was too greatly reduced. From our experience, symptoms of salt-hunger appear when the whey in the mixture is reduced below 300 to 350 c.c. per liter. In the mixture used by Dr. Morse, with very low whey-content, there must have been a

marked salt-hunger in these children. Considered in this light it is self evident that no child could gain on such feeding, and we are very happy that Dr. Morse did not get worse results.

It is not just to compare this food to eiweissmilch which, as described by Dr. L. F. Meyer and myself, contains (without sugar additions) casein 3 per cent., fat 2 to 3 per cent., a little milk-sugar and almost 500 gm. of whey. We are certain that the difference in the results obtained by Dr. Morse and ourselves can be explained on this basis.

Berlin, March, 1912. H. FINKELSTEIN.

PROGRESS IN PEDIATRICS

REVIEW OF THE 1911 STUDIES IN THE NORMAL METABOLISM OF CHILDREN

J. P. SEDGWICK, M.D.
MINNEAPOLIS

Since this subject received its discussion by Langstein and Meyer[1] last year, advance has been made along various lines. Unfortunately this work is not accessible to all, but Grulee's[2] work, now in the press, devotes considerable space to metabolism, and in a measure makes up for this deficiency in our American literature.

Ott and Scott[3] found that extract of the infundibulum starts the flow of milk in about one minute from the beginning of its intravenous injection, and that the excretion reaches its height in four minutes, after which it rapidly falls to normal. They found that the corpus luteum (10 grains), pineal body (5 grains) and the thymus (1 grain) increased the quantity of milk four-fold in five minutes. The ovary minus the corpus luteum had no effect. The amount of butter fat was about the same in the secretion, augmented by the thymus, corpus luteum and infundibulum.

Helbich[4] demonstrated that lactation can be maintained by artificial evacuation of the breast-milk without the physical stimulus of the sucking child. This can be kept up for weeks and months. He showed, moreover, that a poorly secreting breast may have its milk production increased by artificial emptying with a breast-pump. The lactation curve varies very little from that obtained in normal breast feeding.

Studies on virginal lactation made by Pfaundler[5] show the importance of irritation in producing a milk flow. An examination was made of the secretion of a 2-year-old calf which had not been covered or pregnant. The animal had been sucked by other calves, and Pfaundler considered it definitely proved that a fully normal, virginal animal subjected to the irritation of sucking may produce a proper and rather profuse milk

1. Langstein and Meyer: Infant Feeding and Metabolism (Sedgwick Ab.), 1910, Bergman, Wiesbaden.
2. Grulee: Infant Feeding, 1912, Saunders, Philadelphia.
3. Ott, I., and Scott, J. C.: The Action of Animal Extracts upon the Secretion of the Mammary Gland, Arch. Pediat., xxviii, No. 10, p. 1029.
4. Helbich, H.: Is the Physiological Stimulus Necessary for Starting and Maintaining Lactation? Monatschr. f. Kinderh., x, No. 8, p. 391.
5. Pfaundler, M.: Virginal Lactation, Ztschr. f. Kinderh., iii, No. 3, p. 191.

secretion. He reviews the historical cases showing similar results in the human being. He therefore considers that the usual theories concerning normal, virginal lactation require revision.

Basch[6] considers the activity of the breast of the new-born child to be an excellent indicator of the functional capacity of the mother's breast. He gives a very interesting résumé of the literature, as well as clinical and experimental work in supporting his position. He considers it probable that the activating bodies are carried to the fetus from the mother animal by way of the blood.

Talbot[7] found in every instance that the milk-supply of the mothers at the hospital was in excess of the needs of their own babies. The women were paid at the rate of 60 cents a quart for breast-milk.

Frank and Unger[8] give the following conclusions concerning the causes which produce the growth of the mammary gland:

1. Intra-uterine, prepuberty and puberty growth of the breasts are directly dependent on ovarian function (Tandler, Foges).

2. A cyclical change in the virgin breast occurs under the influence of the ovary.

3. Castration does not cause rapid regression of the cyclical breast hyperplasia.

4. No proof has been offered to show that the fetus or placenta directly produces growth of the breast in pregnancy.

5. Evidence points to the fact that the persistent corpus luteum of pregnancy may produce this breast growth.

6. The factors which favor or cause the persistence of the corpus luteum are unknown.

7. Certain evidence (increase of the breast produced by hydatid mole without fetus, chorio-epithelioma) makes it unlikely that the fetus is at any time the controlling factor.

8. Nature's process is more complicated than the simple chemical stimulus assumed by Starling. As yet hyperplasia of the breasts has not been experimentally produced except by parabiosis, which does not explain the stimulus. Possibly the influence of other glands of internal secretion complicates the problem.

9. Milk secretion is no index of quantitative increase in breast tissue.

10. Under physiologic conditions milk secretion sets in when the ovarian influence is removed in the new-born after birth; in the puerpera as the corpus luteum of pregnancy regresses; sometimes postoperatively

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6. Basch, K.: The Mammary Secretion of the Child as an Index of the Secretory Capability of the Mother, München. med. Wchnschr., lviii, No. 43, p. 2266.

7. Talbot, Fritz B.: Two Methods of Obtaining Human Milk for Hospital Use, Boston Med. and Surg. Jour., 1911, clxiv, 304.

8. Frank, Robert T., and Unger, A.: An Experimental Study of the Causes which Produce the Growth of the Mammary Gland, Arch. Int. Med., 1911, vii, 812.

after castration in the virgin (if the breast has been activated by the corpus luteum of menstruation).

Knape[9] found no bad results in feeding children with breast-milk preserved by the addition of perhydrol. Older children are sometimes disturbed by the taste.

Grulee[10] concluded from clinical observations that sodium benzoate to the amount of 2½ to 5 grains in twenty-four hours, given to artificially fed infants ranging in age from a few weeks to almost 2 years produces no recognizable symptoms, even though these children may be suffering from gastro-intestinal disturbances of a serious nature.

Kleinschmidt[11] found that the proteins of cow's milk, casein, albumin and globulin may be differentiated from each other by means of anaphylactic experiments. Albumin from cow's milk and serum from cattle appear to be identical. Casein appears to be more closely allied to the globulin than to the albumin.

A. Filia,[12] as the result of his researches on goats, stated that uric acid and purin bases in small quantity passed through the milk; the administration of food rich in nuclein, injections of lecithin, uric acid given by mouth and by injection, slightly increased the elimination of uric acid and purin bases. He found no difference in the quantity of uric acid and purin bases eliminated by the milk in uremic or healthy women.

Barbier's[13] studies based on a dozen cases show a certain parallelism between the salts and the casein.

Schloss[14] found that the composition of the milk bears no relation to the amount produced in early lactation. Early the amount of calcium is high. This is followed later in human milk by a fairly constant amount of calcium.

According to Plauchu and Rendu[15] the influence of multiparity and age of the nurse, as well as the duration of the breast feeding is variable and not of great importance on the fat content of the milk. The alimentary regimen, as well as galactogogues and menstruation appear to have

9. Knape, Walter: Breast Milk Conserved by Perhydrol, Monatschr. f. Kinderh., 1911, x, No. 6, p. 281.

10. Grulee, Clifford G. and Buhlig, Walter B.: Sodium Benzoate in Artificially-Fed Children, Arch. Pediat., xxviii, No. 10, p. 869.

11. Kleinschmidt, Hans: The Biological Differentiations of the Proteins of Milk, Monatschr. f. Kinderh., x, No. 8, p. 402.

12. Filia, A.: Elimination of Uric Acid by the Milk, Brit. Jour. Child. Dis., 1911, viii, p. 463. Seventh Congress of the Italian Pediatric Society, held at Palermo, April, 1911.

13. Barbier, M.: Session Society of Pediatrics of June 20, 1911, Arch. de méd. d. enf., 1911, xiv, p. 559.

14. Schloss, Ernst: The Chemical Composition of Human Milk, Monatschr. f. Kinderh., x, No. 10, p. 500.

15. Plauchu, E., and Rendu, Robert: Study of Fat in Breast Milk, Arch. de méd. d. enf., 1911, xiv, p. 601.

no effect. On the contrary the more milk a nurse has the less is the butter content. The butter content is higher in the morning than in the evening. The butter content at the beginning of the nursing is higher than at the end of nursing. The smaller of two breasts gives the higher butter content. Too much fat in the milk or too little fat is thought to produce digestive troubles, especially in premature infants.

Autenrieth and Mueller[16] describe colorimetric methods for determination of kreatin and kreatinin based on the principle of the Folin method, and a colorimetric method for sugar, for which they claim the advantage of greater ease and rapidity.

Best[17] found that in the normal person foods prepared in widely different ways were well digested. He considers the importance of the cooking, in as far as the digestion is concerned, to lie mainly in its effect on the appetite and the accompanying psychic secretion and stimulation to motility.

According to Allaria[18] the importance of the chemical reaction of the mouth depends on its influence on the bacterial flora in the cavity, as well as its effect on the diastatic activity of the ferment. The mouth of the new-born is neutral (litmus) with frequent inclination to a slight acidity. In later infancy the contents of the mouth show various chemical reactions when examined with litmus paper. Usually blue litmus paper is turned red. The reaction is rarely neutral or alkaline. There seems to be no apparent relation between the chemical reaction of the contents of the mouth and the secretory irritation. Therefore the saliva may be considered as an almost neutral fluid. The saliva is less often and less actively acid immediately after its secretion than when it has remained in the mouth for some time. The saliva is acid to phenolphthalein.

In another communication[19] he states also that isosmotic food solutions such as milk, or hyposmotic food solutions, arrive in the stomach in a very definite hyposmotic condition; hyperosmotic solutions, on the contrary, go into the stomach in a less hyperosmotic condition, so that the saliva may be considered in the earliest childhood to have an important physical-chemical effect; that is, the regulation, or better, the lowering of the molecular concentration of the food solutions and therefore a decreasing of the degree of osmotic pressure (osmo-regulating or osmo-moderating function of the saliva).

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16. Autenrieth, W., and Funk, Albert: Concerning the Colorimetric Determination of Sugar, Creatin and Creatinin in the Urine, München. med. Wchnschr., vol. lviii, page 899.

17. Best, F.: Concerning the Influence of Preparation of the Food upon its Digestibility, Deutsch. Arch. f. klin. Med., civ, No. 1, 94.

18. Allaria, G. B.: The Chemical Reaction of the Infant's Saliva, Monatschr. f. Kinderh., x, No. 4, p. 179.

19. Allaria, G. B.: Concerning the Action of Saliva in the Beginning of Digestion in the Infant, Jahrb. f. Kinderh., xxiv, No. 3, p. 252.

The arrival of the saliva food-mixture in the stomach causes the immediate secretion of gastric juice rich in hydrochloric acid and of a strong concentration, which has the effect of bringing about an osmotic equilibrium with the blood.

Salle[20] found it possible by means of raising the temperature beyond a certain boundary, in the case of young dogs, to bring about a disease picture characterized by great loss of weight, rise of temperature, diarrhea and vomiting. The examination of the excretory function showed a lessening of the gastric juice, a decrease of the digestive power of the ferment, a lowering of the total acidity and the content of free hydrochloric acid or even the disappearance of the latter.

Bahrdt and Bamberg[21] made examinations on dogs with high duodenal fistula in which the effect of acetic acid and butyric acid was tried on the peristalsis of the stomach and intestine. The quantitative relations were especially considered. It was shown that relatively small amounts of the lower acids, amounts which gradually came into the chyme, were able to hasten the peristalsis. Moreover, they brought about a lengthened closing of the pylorus, a reflex which is to be considered as a protective arrangement for the intestine against the acid-containing food coming out of the stomach. Small amounts which, fed by mouth, brought about no increased stomach peristalsis, increased the peristalsis and brought about vomiting and diarrhea when they passed into the duodenum.

They[22] also found that acetic acid is the most active and the strongest among the substances examined. The higher the acid stands in the series the less active is it in causing peristalsis. It seems possible that the organic acids which are formed in the stomach normally may, in case of pathological increase of collection, be the cause of acute dyspepsia.

Huldschinsky[23] gives results which are to be considered in relation to normal digestion only:

1. In feeding with human milk the stomach contains only very small amounts of volatile acids. When cow's milk is fed it contains from three to six times as much.

2. The amounts of volatile fatty acids found in the stomach when cow's milk is fed corresponds with the amount of fat contained in the food. Protein and sugar, however, have no influence.

3. The formation of the volatile acids in the stomach of the normal infant can only be explained by the fermentative splitting of the glycerids

20. Salle, V.: The Effect of the Air Temperature upon the Secretory Activity of the Stomach, Jahrb. f. Kinderh., xxiv, No. 6, p. 697.

21. Bahrdt, H., and Bamberg, H.: Animal Experiments upon the Action of the Lower Fatty Acids upon Duodenal Peristalsis, Ztschr. f. Kinderh., 1911, iii, 313.

22. Bahrdt, H., and Bamberg, H.: Animal Experiments upon the Action of the Lower Fatty Acids upon Duodenal Peristalsis, Ztschr. f. Kinderh., 1911, iii, 350.

23. Huldschinsky, K.: Concerning the Volatile Fatty Acids in the Stomach Content of Normal Infants, Ztschr. f. Kinderh., 1911, iii, 366.

of these acids. The kind of lower fatty acid which is formed corresponds with the preformed acids in the milk fat.

Acetic acid is formed in small amounts only in the normal infant stomach. It appears possible that through a pathologic increase of the amount of cow's milk in the stomach there may arise acids which in themselves are less toxic; that is, butyric, caproic and caprylic acids, but which in greater amounts increase the peristalsis and cause acute dyspepsia.

According to Schmidt,[24] pieces of vegetable tissue which are left two to four hours in the incubator with a pepsin-hydrochloric acid solution and then placed in a solution of pancreatic ferments show marked softening so that a slight pressure of the finger or even a shaking of the test-tube change them into a gruel-like mass. This influence of the stomach on vegetables must be considered as a chemical action. In the past the breaking-up function of the stomach has been given too little attention in comparison with the dissolving function.

Davidsohn[25] states that pepsin is found in the infant stomach, but exerts no digestive action. He considers the coagulating ferment and the proteolytic ferment one.

Kleiner[26] found that the contents of the stomach and small intestine of normal rabbits, kept on a diet poor in easily convertible carbohydrates, when removed immediately after killing the animal, usually contain a very small but measurable amount of dextrose. A preceding nephrectomy does not increase the amounts of the dextrose in these viscera.

Sedgwick and Schlutz[27] found that a preliminary digestion of fat with gastric lipase increased the action of pancreatin on emulsified fat. Whether the effect shown was a summation or activation was not proved, but they concluded that the function of the gastric lipase does not end when the stomach contents pass the pylorus and are neutralized in the duodenum.

Cowie and Lyon[28] found that the pyloric opening and closing can be easily demonstrated in infants' stomachs. Evacuation of the stomach was delayed by experimental acidity (sustained duodenal closing reflex), as well as in experimental alkalinity (delayed pyloric opening reflex). Free acid was not found necessary for pyloric opening in the infant. In hyper-

24. Schmidt, Adolf: Concerning the Comminuting Function of the Stomach in the Normal and Diseased Stomach, Deutsche med. Wchnschr., 1911, xxxvii, No. 10, 435.

25. Davidsohn, Heinrich: Contribution to the Chemistry of the Infant Stomach, Ztschr. f. Kinderh., ii, No. 5, p. 420.

26. Kleiner, Israel: The Excretion of Dextrose in the Stomach and the Small Intestine, Jour. Exper. Med., 1911, xiv, 274.

27. Sedgwick, J. P., and Schlutz, F. W.: Relationship of Gastric to Pancreatic Fat Digestion in Infants, Am. Jour. Dis. Child., 1911, ii, 243.

28. Cowie and Lyon: Further Observations of the Acid Control of the Pylorus in Infants, Am. Jour. Dis. Child., 1911, ii, 252.

secretion in infants, protein-containing food appeared to leave the stomach more readily than water because of its binding property for acid.

Klotz[29] finds the acid formation in pancreatic digestion weakest with wheat. It is increased with rye, still more with barley, and to the highest degree with oats.

His results show that the bacterial content in the stool of the breast-fed child corresponds in general to that which we find in the bottle-fed child which is receiving two carbohydrates. The different forms of sugar influence the fermentative process with different intensity. Especially aggravating in effect are the starches, and among them more noticeable energetically, barley and oatmeal. It is possible to increase the number of bacteria in the feces to a maximum by a pre-arranged variation of the carbohydrates. Bacteria appear in the infant to represent from 30 to 36 per cent. of the dry fecal substance. It seems, moreover, not improbable that when an increase of the bacteria is not found when we are ministering high molecular carbohydrates, that a disturbance in the working together of the intestinal enzymes and intestinal bacteria is indicated.

Edelstein[30] says:

1. Dyspeptic and normal stools of breast-fed children show acetic acid as the chief constituent among the volatile fatty acids. The amount of butyric acid may be as high as 0.06 per cent. in the normal stool from the breast-fed child. In dyspeptic cases the stools may show small quantities of caprylic and formic acid.

2. The volatile fatty acids in the stomach contents of children fed with full cow's milk consists of caprylic, caproic, butyric and a little acetic acids. Caprylic and butyric acids are present in like amounts.

3. In buttermilk, according to Koppe (Vibelmilch), and in sour milk which has stood for two days, the volatile fatty acids, principally acetic acid, are present. In the sour milk up to 0.05 per cent. and in the Vibelmilch up to 0.03 per cent.; that is, acetic acid with a little butyric acid.

Bauer[31] confirms the work of Talbot on curds.

Brennemann[32] says that in all his experience he has never seen a hard curd unless a considerable amount of raw milk casein was given, and has never seen hard curds persist or occur when boiled milk, no matter in what amount, was given. He considers the difference in the action of rennin on raw and boiled milk as significant in this connection.

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29. Klotz, M.: Studies on Starch Digestion, Jahrb. f. Kinderh., lxxiii, 391.

30. Edelstein, F., and Csonka, F.: The Qualitative (Quantitative) Determination of Volatile Fatty Acids, Ztschr. f. Kinderh., 1911, iii, 321.

31. Bauer, J.: Concerning Casein Curds in Infants' Stools, Monatschr. f. Kinderh., 1911, x, No. 5, p. 239.

32. Brennemann, J.: Etiology and Nature of Hard Curds in Infants' Stools, Am. Jour. Dis. Child., 1911, i, 341.

While Ibrahim[33] confirms the findings of Wernstedt and Talbot, he also considers the casein "curds" more common in uncooked milk.

Pfaundler[34] calls attention to the fact that Talbot's biologic method does not show a protein body of definite chemical constitution, but only certain groups attached to the protein bodies. Therefore, strictly considered, the experiments of Uffenheimer and of Talbot and others show only the presence of such foreign radicals, not, however, the presence of casein or derivatives of the same in the infant's stool. He grants freely, however, that it is probably a casein derivative.

Stolte[35] considers that one common principle is fundamental in the methods of feeding which give good results, and that is the formation of soap or solid stools.

Saito[36] says the fat content of the feces of the infant is almost constant, either by natural or by artificial feeding; that is, it makes up about 20 per cent. of the salt constituent of the stools. Fat amounts to about 96 per cent. in the new-born child; especially in artificial feeding the fat content of the feces is usually higher and may go at times up to 40 per cent. or even more. In dyspepsia the fat content seems to be higher. Atrophic children always show a higher fat content, which becomes lower in convalescence. The feces of the new-born child, as in dyspepsia, are especially rich in oleic acid, which decreases in amount as the child grows, or in convalescence. The so-called "curds" in the feces are chiefly solid soap compounds of the fatty acids; 0.03 per cent. of the dried substance of the feces is made up of cholesterin and contains no saponifiable substances. In a rough manner the composition of the fat in the feces may be recognized by microchemical means. If a drop of concentrated sulphuric acid be put on a small piece of feces, numerous fatty acid crystals appear in the place of the soaps, and the individual fat drops are still plainer under the microscope.

Engel and Turnau[37] state that urines of breast-fed children react positively; that is, they show a black precipitate, while the urines of artificially fed children give a negative reaction when treated as follows: 15 or 20 drops (about 1 c.c.) of a 2 per cent. silver nitrate solution are added to 5 c.c. of urine. This is allowed to stand quietly for about ten minutes.

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33. Ibrahim, J.: Curds in Connection with the Feeding of Raw Milk, Monatschr. f. Kinderh., 1911, x, No. 2, p. 55.

34. Pfaundler: Discussion of Uffenheimer's Paper Before the Münchener Gesellschaft für Kinderheilkunde, Jahrb. f. Kinderh., lxxiv, No. 1, p. 82.

35. Stolte, Karl: Concerning the Conditions Requisite for Formation of Solid Stools of Infants, Jahrb. f. Kinderh., lxxiv, No. 4, p. 367.

36. Saito, Hideo: On the Fat Content of the Feces of Infants, Jahrb. f. Kinderh., lxxviii, 222.

37. St. Engel and Turnau, L.: A Reaction Given by the Urines of Breast-Fed Children, Berl. klin. Wchnschr., 1911, xviii, No. 1, p. 18.

Boschan[38] thinks that the Engel and Turnau urine reaction does not depend on whether the child is fed with breast or cow's milk, but on the chlorid, phosphate and carbonate content of the urine.

The discoverers of the reaction state, however, that they had recognized the fact all along that the chlorin is of great importance in this silver nitrate reaction. They state that on the basis of numerous chlorin determinations in the urine of breast-fed children, they have found that this amount of silver nitrate is sufficient to oversaturate the maximum chlorin content in 5 c.c. of the urine from breast-fed children. Naturally the key to the reaction lies in these quantitative data. If the amount of silver nitrate used with the urines of the infants fed with cow's milk is increased above the saturation point, a discoloration is also produced. This, however, is different from that of the urine of breast-fed children which shows from a yellowish to a reddish brown. If there is any doubt it is advisable to compare the color with the reaction from a breast-fed normal child.

Woodyatt[39] and Helmholz found the losses in "The Use of Blood Charcoal as a Clearing Agent for Urine Containing Glucose" were not constant even in the same urines, but varied widely with the weight of charcoal used, the amount of shaking and the duration of contact.

Different samples, however, acting in divergently opposite ways, make it necessary that, before any conclusions be drawn from measurements made by the Bang and Bohmannsson method, the individual sample of charcoal which is used shall be thoroughly tested in control experiments.

Amberg[40] and Knox consider it probable that a very appreciable part of the nitrogen contained in the urine of the normal breast-fed infant is excreted in the form of hippuric acid.

Fleischner[41] found that meat given twice daily to children of 6 years gives rise to a decided increase in the indicanuria and is a cause of pronounced intestinal putrefaction. This was found even more markedly the case in 3-year-old children. Meat given twice daily to 9- and 12-year-old children did not cause an increase of indicanuria. He concludes that the giving of meat more than once daily to children under 9 years of age is inadvisable.

Reuss[42] states that indican is often found in the urine of breast-fed children during the first weeks of life, sometimes in large quantities,

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38. Boschan, F.: The Character of the Engel-Turnau Reaction, Berl. klin. Wchnschr., 1911, xviii, No. 7, p. 302.

39. Woodyatt, R. T., and Helmholz, H. F.: The Use of Blood Charcoal as a Clearing Agent For Urine Containing Glucose, Arch. Int. Med., 1911, vii, 598.

40. Amberg, Samuel, and Knox, J. H. Mason: Hippuric Acid in the Urine of Normal Breast-Fed Infants, AM. JOUR. DIS. CHILD., 1911, ii, 248.

41. Fleischner, Charles: The Relationship of Meat Ingestion to Indicanuria in Children, AM. JOUR. DIS. CHILD., 1911, ii, 262.

42. Reuss, A.: Indicanuria in the New-Born, Ztschr. f. Kinderh., iii, No. 1, p. 12.

without its being possible to demonstrate chemically any pathologic phenomenon. It is found with good nutrition and with stationary weight, with frequent bowel movements and where there is an inclination to constipation. Indicanuria is usually lacking on the first day, is rare on the second day, and is found more commonly and intensively on the third and fourth days. Moreover, during the following days its appearance is not rare.

The summary of Hamill and Blackfan's study[43] of the albumin in the urines of normal children is too valuable to omit:

1. There has been no relationship between the specific gravity and the form or amount of albumin.

2. The reaction has had no influence on the production of albumin.

3. Sugar, acetone and diacetic acid were never found. They may, therefore, be considered as having no bearing on the production of albumin.

4. Indican, phenol and urobilinogen when present were usually associated with albumin, but albumin was sometimes absent when they were all present, and the amount was never greater when associated with them than it was in the cases in which they were absent.

5. Crystals, when present in amounts, such as are occasionally found in normal children, are in no way responsible for the associated albumin.

6. The milk disturbances of the intestinal digestion, as shown by the examination of the stools, were not sufficient to account for the occurrence of albumin.

7. The blood-pressure was within the normal range in all cases, and therefore did not influence the albumin output.

8. The albumin elimination was the same on mixed and exclusive milk diets.

9. They found no children in whom the albumin excretion corresponded to the requirements for postural or orthostatic albuminuria, a rather surprising result in view of the frequency with which this condition is supposed to occur.

10. Thirty-two and one-half per cent. of the children showed occasional hyaline casts and cylindroids in their urines. They do not consider their "occasional presence" as indicative of a lesion of the kidneys, but rather as suggesting a temporary overtaxation of the kidneys resulting from variations in the habits of life of the individuals which are too slight to be recognized.

11. Eighty-eight and seven-tenths per cent. of the urines of these 124 children showed albumin, 27.4 per cent. showing serum-albumin alone, and in combination; and 85.4 per cent. an albuminous body precip-

43. Hamill, Samuel McClintock, and Blackfan, Kenneth D.: Am. Jour. Dis. Child., 1911, i, 139.

itated by acetic acid in the cold. These two albumins were nearly always present in very slight traces, occasionally in slight traces and rarely in traces.

In thirty-eight children the twenty-four-hour specimens showed nucleo-albumin in all but one, and in this case samples examined over prolonged periods of time showed nucleo-albumin frequently.

In these thirty-eight children, the percentage of serum-albumin was very much higher (42.1 per cent.) than in the total number of cases examined. They believe therefore that it is possible to demonstrate in the urine of every presumably healthy child traces of an albuminous body precipitated by acetic acid. Consequently this substance must be regarded as an exceedingly common, if not constant manifestation in the urine of children up to 14 years of age, and as of no clinical significance.

They do not believe that serum-albumin in the amounts in which it appears in these children indicates a diseased condition of the kidneys any more than does the presence of occasional hyaline casts and cylindroids, and that its etiology may be considered the same as that given for these former elements.

Simon's[44] statement that the urines of new-born infants on which he worked did not give creatinin reactions at the dilutions recommended by Folin does not agree with the results of Sedgwick, who found reactions of 10-250 to 10-500 dilutions to be common. All of Sedgwick's infants were at the mothers' breasts and were usually given no other fluid, which could dilute the urine. Simon found that new-born infants have a high nitrogen excretion during the first week which sinks to the normal proportions later. He found that the new-born child (during the first few days of life) excretes unsplit polypeptids up to 12 per cent.

Hadlich[45] finds that the amino-acid content of the urine of older children corresponds to that in adults. Diseases with fever do not generally increase it. It is, however, increased by severe enteritis. In the infant the value is generally raised, but apparently is not dependent on the condition of the child or the food or character of the stool. There is one condition especially, however, in which this does not hold, and that is alimentary intoxication. This corresponds to the views of Finkelstein in that it is in this condition that the oxidation capability of the organism is disturbed. The presence of uric acid in the urine indicates an incapacity of the organism to catabolize the amino-acids brought to it from the intestine further, to the point of ammonia, and by this means prepare them for the formation of urea.

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44. Simon, S.: The Nitrogen Partition in the Urine of the New-Born, Ztschr. f. Kinderh., 1911, ii, 5.

45. Hadlich, Richard, and Grosser, Paul: The Amino-Acid Content of the Urine of Children and Infants, Jahrb. f. Kinderh., lxxiii, 421.

Reuss[46] finds that the urines of newly born children contain glycocoll as a normal constituent.

According to Birk,[47] in the artificially fed new-born child about one-half of the nitrogen introduced is excreted in the urine. In the naturally fed child only one-sixth to one-seventh of the nitrogen is given off. From the above a fundamental difference between the artificial and natural feeding of the new-born child is shown. It is almost like a law to be observed that the longer the period of underfeeding continues, just so much more intensive are the symptoms of exudative diathesis which develop later.

The hypodermic injection of urine in the lower animals causes the appearance of hemolytic precipitates, and complement-binding antibodies in their serum. The precipitation is always stronger with normal than with protein-containing urine. On the contrary the protein-containing urine produces more complement binding.

The apparatus designed by Lawrence[48] for collecting urine in female infants, for use in the Boston Floating Hospital, has been found very valuable by the reviewer. It consists of an adhesive plaster strap one-half to 2 inches wide and 4 to 6 inches long, split at one end, thus forming two tails to be directed backward and outward over the buttocks. An elliptical opening is cut through the strap and through this opening the thumb of an old rubber glove is passed and turned down on the adhesive surface of the strap. The tip of the thumb is then cut out and a test-tube inserted in the opening. The test-tube may be wrapped with adhesive, if desired, to avoid slipping. The opening in the adhesive, armed with the rubber thumb, is placed with the adhesive portion toward the body so that the opening is over the vulva. The urine is then passed directly into the test-tube.

For collecting twenty-four-hour specimens the test-tube is replaced by a piece of glass tubing which is connected by a rubber piping with a bottle placed inside the bed.

Interesting apparatus for the collection of excreta is described by Dubois,[49] as well as by Howland and Cook.[50]

The minimum, as well as the maximum, blood-pressure increases with the age of the child, according to Sahle,[51] and is, moreover, dependent

46. Reuss, A.: Concerning the Presence of Glycocoll in the Urine of the New-Born Infant, Ztschr. f. Kinderh., iii, No. 3, p. 286.

47. Birk, W.: Contribution to Physiology of the Newborn Child, Monatschr. f. Kinderh., 1911, No. 1, p. 5.

48. Lawrence, Charles H.: A Method of Collecting Urine from Female Infants, Boston Med. and Surg. Jour., 1911, clxiv, 309.

49. Dubois, Eugene F.: An Apparatus for the Collection of the Excreta of Infants, Am. Jour. Dis. Child., 1911, ii, 415.

50. Howland and Koch: A Metabolism Bed for Infants, Am. Jour. Dis. Child., 1911, ii, 419.

51. Salle, V.: Blood-Pressure in Children, Jahrb. f. Kinderh., lxxiii, 273.

within certain boundaries, on the weight and size of the child; also the height of the amplitude becomes greater with increasing age. Deviations from the average are shown in lymphatism (lower), neuropathy and nephritis (higher).

Emerich Rusz[52] finds the absolute values of the viscosity of the blood and refraction of the plasma to vary within wide boundaries according to the condition of the infant. The viscosity rises during the first days of life, then sinks sharply. This decrease then goes on more slowly through the whole first year. The refraction rises sharply also during the first days of life. There is then under normal conditions a decrease up to the fourth or eighth day after which it begins to rise again strongly. This rise continues then slower and slower through the whole first year of life. The day's variations of the refraction and viscosity of the blood are most marked from morning to midday. The decrease begins at midday and goes on until evening. The cause of the high morning value is the concentration of the blood during the eight hours of the night fast. The influence of the taking of food on the refraction and viscosity of the blood appears to differ with the condition of the child. The normal child does not seem to be affected by the food unless it causes a decrease. When the child is not progressing an increase occurs.

Miss Michael's[53] work confirms the blood-pressure results of Frau Wolfensohn-Kriss, as she found the pressure to increase apparently with height and weight, as well as age. She found, however, on application of Sallom's formula, thus obtaining the blood-pressure per square millimeter, that the actual blood-pressure of a 40-pound 5-year-old child and that of an 80-pound 15-year-old were practically the same.

Mills'[54] summary and conclusions concerning the utilization of fats and oils given subcutaneously were as follows:

"1. Olive, peanut, cocoanut, sesame, cotton-seed, lard-oils, unsalted butter-fat and lard may be given hypodermically and over a considerable period without local irritation, provided aseptic care is used, and no constitutional disturbance occurs provided precautions are used to prevent injection into the blood-stream.

2. Emulsions of these oils made with 3 to 5 per cent. of egg lecithin and water are permanent, and cause no irritation if given subcutaneously.

3. Oils and fats given subcutaneously are absorbed by means of the lymphatic system and eventually reach the thoracic duct.

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52. Rusz, Emerich: Physiological Variations in the Viscosity and Refraction of the Blood of Infants, Monatschr. f. Kinderh., 1911, x, No. 7, p. 36.

53. Michael, May: A Study of Blood-Pressure in Normal Children, Am. Jour. Dis. Child., 1911, i, 272.

54. Mills, Lloyd, H.: The Utilization of Fats and Oils Given Subcutaneously, Arch. Int. Med., 1911, vii, 694.

4. Lymphatic vessels and glands in contact with and transmitting oil for any length of time become hypertrophied and are thus better able to carry oil.

5. The amount of absorption of plain oil from the subcutaneous tissues after injection during starvation is so small as to be negligible. Emulsified oils and fats injected during starvation are absorbed in amounts sufficient to furnish from one-half to two-thirds of the full calorific requirement of the animals injected.

6. Oils and fats so injected and absorbed have no more influence on the destruction of protein in starvation than has fat given alone by mouth.

7. Plain oils injected subcutaneously under conditions of low protein ingestion are little, if any, better absorbed than when similarly given during starvation. Emulsified oils injected under these conditions are absorbed quite as well as similar oils given to starving animals.

8. Plain and emulsified oils are absorbed about equally well when the animals injected are given a plentiful supply of protein in their food. This probably furnishes the large quantity of lipolytic enzymes necessary for body action on plain oil.

9. The injection of oils subjected to lipolysis causes death, which is due apparently to the production of oleic or other acids, with the possible formation of toxic quantities of soaps.

10. Oil absorbed from the tissues after subcutaneous injection is (*a*) burned in the body for the production of heat and energy, thus sparing the body fat; (*b*) retained as such within the organism; or (*c*) possibly converted into body fat by reconstruction in the liver, from which it may be sent for storage to the various fat depositories, after which it is drawn on as needed. Proof of this last proposition is lacking.

11. It seems likely, from comparative examination of the iodin indices of the ether extracts of visceral and adipose tissue, that the actively functioning viscera use oil and fat absorbed after subcutaneous injection for the direct performance of their functions, and that the storage of the foreign fat given in excess of the nutritive requirement takes place principally in the subcutaneous tissue, liver and lungs, to a small extent in the kidneys and spleen, while the pancreas and stomach and intestines are practically uninfluenced.

12. This demonstration that after injection under suitable conditions oils can be absorbed to an amount capable of covering so large a proportion of the calorific requirement suggests the application of such injections to the treatment of wasting diseases, to the cachectic conditions associated with imperfect metabolic processes and especially to tuberculosis, in which the intolerance to fats is almost symptomatic."

The pediatrist will at once recognize the value of a perfected method of hypodermic feeding.

Carpenter[55] found the energy metabolism of the new-born child expressed per unit of weight, obtained by subtracting the metabolism of the mother alone from that of mother and child together, to be two and a half times that of the mother. Expressed per unit of surface, the energy metabolism of the new-born child was not found greater than that of the nursing mother, but higher than that of a woman in complete sexual rest.

The energy quotient among the new-born fluctuates within wide range according to Samelson[56] so that it would probably be well in place of the former ideas, to set it at from 115 to 150 calories.

Hess[57] says that the energy quotient needed by premature and underweight infants averages between 115 and 170 calories when below 1,500 gm. in weight; when over 1,500 gm. the requirement is not so high — between 100 and 132 calories.

Niemann[58] considers the water balance the most important of his metabolism experiences. The question, "What does the difference in weight of the body on the scales mean? Is it body substances or is it water?" he believes one of greatest importance for the understanding of the condition of the child and for the condition of the metabolism processes of the infant. And this is one of the questions which we find most difficult. We have to consider the water introduced and the water formed by oxidation. Niemann believes that in weaker, normal infants, as one of his children was, water is lost with the taking on of body substance and also that when the child is brought under favorable conditions of nourishment and forced to oxidize body substance, its behavior toward the water does not change and at any rate no water is added. Under no conditions did there appear to be a tendency to putting on water in the case of simple body increase. In the case of very dry air much water was lost by the respiration. He found that with approximately the same body weight an artificially nourished child gives off considerably more carbon dioxid and water per square meter of surface than one naturally fed. On the contrary, two artificially fed children in spite of marked differences in weight gave off almost the same amount.

The daily capacity per square meter of surface was 1,347 calories. Four of his very interesting tables may be included:

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55. Carpenter, Thorne M., and Murlin, John R.: The Energy Metabolism of Mother and Child Just Before and Just After Birth, Arch. Int. Med., 1911, vii, 184.

56. Samelson, S.: The Contribution to the Physiology of the Feeding of Premature Infants, Ztschr. f. Kinderh., 1911, ii, 31.

57. Hess, J. H.: A Study of the Pyloric Needs of Premature Infants, Am. Jour. Dis. Child., 1911, ii, 302.

58. Niemann, Albert: The Total Metabolism of an Artificially-Fed Infant, Jahrb. f. Kinderh., 1911, lxxiv, No. 6, pp. 22, 650.

TABLE 1.—SHOWING WATER BALANCE IN AN INFANT DURING A SEVEN-DAY OBSERVATION

Water Excreted	1st Day	2nd Day	3rd Day	4th Day	5th Day	6th Day	7th Day
Through respiration	304.8	288.0	278.4	288.0	300.0	254.4	328.8
Through sweat	6.5	.....	.....	3.0	5.0	1.0	16.9
Through urine	500.0	470.0	490.0	445.0	510.0	460.0	405.0
Through feces	28.0	28.0	28.0	28.0	28.0	28.0	28.0
Total water excretion	839.3	786.0	796.4	744.0	843.0	743.4	778.7
Water given	693.8	693.8	693.8	668.0	668.0	689.0	745.0
Balance	—145.5	—92.2	—102.6	—76.0	—175.0	—54.4	—33.7

TABLE 2.—ESTIMATE OF CARBON BALANCE IN AN INFANT DURING A SIX-DAY OBSERVATION

Carbon Excretion	— Sum of Days — 1-3	4-6	24 Hr. Average for Days 1-6
Through respiration	151.3	148.0	49.9
Through urine	6.1	4.0	1.7
Through feces	8.1	8.0	2.7
Total C. excretion	165.5	160.1	54.3
Carbon taken in	224.7	168.8	68.6
Carbon balance	+59.2	+26.7	+14.3

TABLE 3.—NITROGEN BALANCE DURING A SIX-DAY OBSERVATION ON AN INFANT

Nitrogen Excretion	— Sum of Days — 1-3	4-6	24 Hr. Average for Days 1-6
In the urine	9.12	9.68	3.13
In the feces	1.20	1.20	0.40
Total Nitrogen excretion	10.32	10.88	3.53
Nitrogen taken in	13.25	14.19	4.57
Nitrogen balance	+2.93	+3.31	—1.04

Per kilogram of weight and day the following values are given:

Calorie intake (net)	114 cal.
Taken on	29 cal.
Heat formation	85 cal.

In this case the daily production of heat per square meter of surface amounted to 1,297 calories.

Salge[59] believes it important for clinical purposes that the young infant can regulate the osmotic pressure, or better, the physiologic composition of its body fluids, but imperfectly. The physiologic insufficiency of regulation, as it is seen in the young infant, gives the basis for various pathologic conditions.

59. Salge, B.: Concerning the Physical Properties of the Blood of Infants, Ztschr. f. Kinderh., 1911, ii, No. 5, p. 347.

Adler and Blake's[60] conclusions are as follows:

1. The amount of base excreted in the urine in acidosis, while following in general the variations in the amount of ammonia, may show differences which are of importance in estimating the power of the organism to resist intoxication.

2. Whether the amount of base excreted varies with the amount of ammonia or not, it is important to determine both quantities as they represent two distinct mechanisms of defense.

Rulon and Hawk[61] found in their studies on water-drinking that in two of the experiments there was a pronounced increase in the output of chlorids on the days of added water intake with a return to normal during the final period. This augmented excretion of chlorids they interpreted as indicating that the large volume of water ingested during this period markedly stimulated the secretion of gastric juice. The excess hydrochloric acid, thus passed into the intestine, was reabsorbed and appeared, at least in part, in the urine as ammonium chlorid. The main bulk of the increase in the chlorid excretion they believe to have originated in this way. The flushing of the tissues and the stimulation of protein catabolism brought about by the copious water-drinking may have been contributing forces in causing the increased output of chlorid observed.

Mendelssohn[62] states that the power of temperature regulation is usually insufficient in very small and weak children. The capacity of regulation is gradually acquired during the first months of life and may be again lost in severe diseases such as intoxication and atrophy.

Koeppe[63] says that marked increases of weight above the normal may owe their occurrence to restitution or water retention. The phosphate content of the urine he found dependent on the phosphate content of the food. When he gave salt in the infant's food he noticed a rise in temperature, increase of weight and a higher amount of phosphorus pentoxid excreted in the urine. He noticed also in the infant that when it had fever and at the same time was receiving the usual amount of food, the increase of weight which depended on the water retention, was accompanied with an increased excretion of phosphorus pentoxid in the urine. In analogy with the adult, he concludes that this is dependent on a chlorin retention.

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60. Adler, Herman M., and Blake, Gerald: The Retention of Alkali by the Kidney with Special Reference to Acidosis, Arch. Int. Med., 1911, vii, 479.

61. Rulon, S. A., Jr., and Hawk, P. B.: Studies on Water-Drinking, Arch. Int. Med., 1911, vii, p. 536.

62. Mendelssohn, A.: Observations Concerning the Surface Temperature of Infants, Ztschr. f. Kinderh., iii, No. 3, p. 292.

63. Koeppe, Hans: Studies in the Mineral Metabolism, Jahrb. f. Kinderh., lxxiii, 9.

Those who are further interested in the important field of salt metabolism may read Schloss'[64] review with profit.

Wiggers[65] gives a résumé of the present understanding of the physiology of the pituitary gland.

Schloss[66] and Crawford found the uric acid output in new-born infants both absolutely and relatively high. In the case in which the umbilical cord was ligated late they found the output of uric acid greater during the second and third days than in those cases in which the cord was ligated immediately after birth. They found the total phosphorus excretion high during the first three days and then it showed a marked diminution. They found a marked retention of the phosphorus in the new-born infant, as well as a moderate nitrogen retention during the first three days. The results show an inverse ratio between the leukocyte count and the elimination of phosphorus and uric acid during the first few days of life. They believe that the parallelism between the excretion of uric acid and phosphorus during the first three days would seem to indicate a common origin from cell nuclei.

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64. Schloss, Ernst: Progress in the Field of Mineral Metabolism of Infants During the Past Three Years, Jahrb. f. Kinderh., xxiv, No. 1, p. 91.

65. Wiggers, Carl J.: The Physiology of the Pituitary Gland and the Actions of Its Extracts, Am. Jour. Med. Sc., 1911, cixl, 502.

66. Schloss and Crawford: The Metabolism of Nitrogen, Phosphorus and the Purin Substances in the New-Born; With Special Reference to the Causation of the Uric Acid Infarcts of the Kidney, AM. JOUR. DIS. CHILD., i, 203.

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